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Health Patterns for American Indians and Alaska Native Children:

Evidence from a Nationally Representative Sample

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Health Patterns for American Indians and Alaska Native Children:

Evidence from a Nationally Representative Sample

by

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Dedication

For my mother.

Without you, I would never have had the confidence to do…anything.

I may be your Sunshine Girl, but you are my eternal source of light.

For my father.

Dad, you did not live to see another “doctor” in the family,

but I finally lived up to your name. We never quite mended our fences,

but at least we bought the lumber and nails.

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Health Patterns for American Indians and Alaska Native Children: Evidence from a Nationally Representative Sample

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The objective of this research was to evaluate the risks and protections for selected child health outcomes, especially among American Indians and Alaska Natives (AI/ANs), in the United States, 1997-2003. These outcomes were asthma, three or more ear infections, health limitation, and injury and were selected because they were available in the selected data and because they are salient for the target population, AI/ANs. The methods employed a national data set, the National Health Interview Survey 1997-2003 (N=67,903) from which the child sample, adult sample, person, family, and injury files were drawn. Variables used to predict the selected outcomes were categorized as child characteristics, socioeconomic factors, and environmental factors (parental health behaviors). Both race-inclusive and race-specific logistic regression models were estimated to predict child health. As the unit of analysis, only children whose responding adult was a parent were included. Children of pregnant parents were excluded to preserve adult body mass index. Overall, the results indicated the child characteristics that affected the selected outcomes were race-ethnicity, sex, age, and birthweight. The socioeconomic factors that consistently presented risk were having one parent in the

home and having government insurance. The parental health behaviors robust across the varied outcomes were parental smoking, parental weight status, parent self-rated health, and the accumulation of negative parental behaviors. Children from the AI/AN population were at significantly greater risk for asthma in the full model, with an odds ratio of 1.37 compared to non-Hispanic Whites, the reference group. The children classified as AI/AN also had an increased risk for recurrent ear infections; however these results were not significant. There were no significant differences in the odds of AI/ANs having a health limitation or an injury compared to the referent. In conclusion, American Indian and Alaska Native children were at greater risk for two of the selected health outcomes, asthma and recurrent ear infections, compared to non-Hispanic Whites. Small cell sizes prevented stable estimates in race-specific models for the target population.

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Chapter 1, Introduction

1.1 RESEARCH PROBLEM AND OBJECTIVES

This research is focused on the American Indian and Alaska Native (AI/AN) population and public health. The American Indian and Alaska Native population comprise a very small group (less that one percent of the U.S. total population) and therefore do not capture the interest of many researchers or public health officials on a national level.[1] However, they are an underprivileged group, with severe health disadvantages compared to other race-ethnic groups, and thus, a population in need of considerable research attention (Murray et al 2006, Castor et al 2006, Matthews, Menacker & MacDorman 2003, Snipp 1999, Sandefuer 1989). Thus far, much of the public health attention given to AI/ANs has been at the tribal level (E.g. Farmer, Bell & Stark 2005, Pollex et al 2005, Scavini et al 2005, Moss et al 2004, Epple et al 2003). Even at this level, research has largely focused on adult health, leaving determinants of child health outcomes unexplored. Thus, this research addresses a *problem* in current literature and tries to fill a major gap in health research involving AI/AN children.

In studying the health of AI/ANs, the question inevitable arises, "Why do AI/ANs remain disadvantaged in terms of health?" There are several, possible explanations. These include their position in social structure (historically and currently), cultural practices, social pathologies, and intergenerational transition of inequality. Their historical and current position in society is one of forced assimilation, segregation, and marginalization. Cultural practices that may contribute to poor health include a high fat diet and lack of exercise. Many consider to social pathologies exhibited by AI/ANs a

[1] A total of only 0.9% if the U.S. population reported their race as American Indian or Alaska Native (AI/AN) for the 2000 Census. Another 0.6% reported their race as AI/AN as well as one or more other races (Ugonwole 2002).

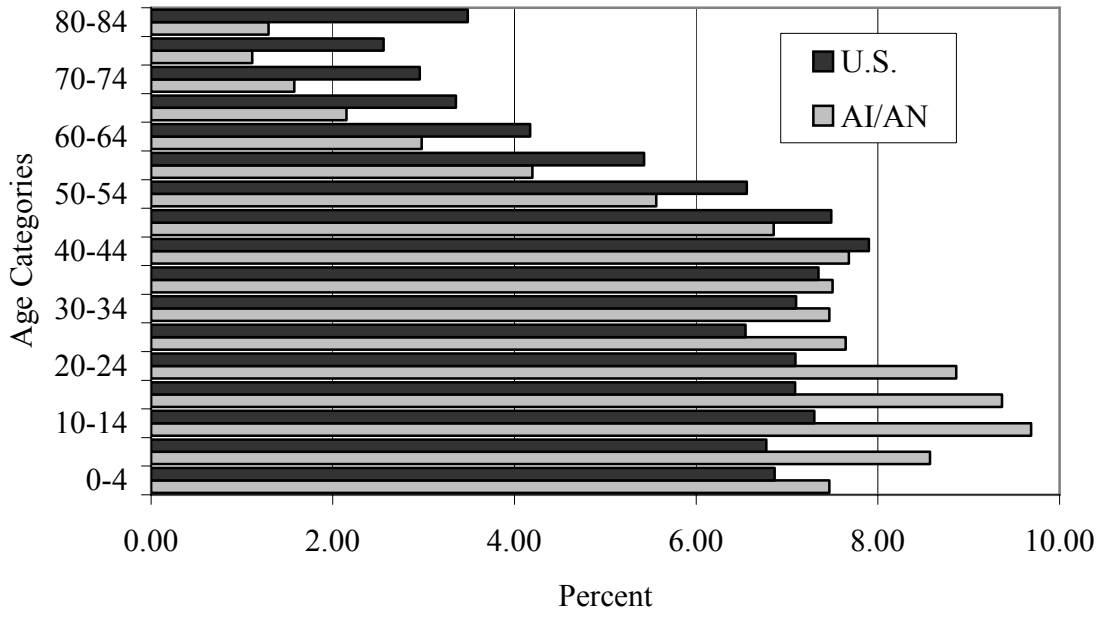
result of their historical treatment in society. These pathologies include alcohol or substance abuse, homicide, suicide, and violence. American Indians and Alaska Natives also have little resources to pass on to their children, sharing with them a deficit in social capital rather than a surplus of social capital.

Research on socioeconomic and environmental characteristics and how these affect health outcomes is well established in social science (Boardman 2004, Mirowsky & Ross 2000, Robert 1999, Wallace & Wallace 1997, Link & Phelan 1995, Williams & Collins 1995, MacIntyre, MacIver & Sooman 1993, Williams 1990). "Environment" in this context refers to the child's social and family environment, not his or her physical environment. This work follows that same line of inquiry. Specifically, this research will explore how sociodemographic variables, parental characteristics and birth outcomes are related to particular child health outcomes: asthma, ear infections, health limitation, and injury. This research agenda will explore both protective and risk factors for each outcome. The terms "protective" or "protection" in this project refer to compensatory protection. These terms are often used to identify risk factors that reduce the impact of other risk factors (to identify interaction terms). This is not how these terms are used in this research. Protective variables here are those that offset poor health outcomes in and of themselves. In addition, this project will seek to determine whether or not the mechanics of being disadvantaged have the same effect on AI/AN health as they do for other race-ethnic groups.

Investigating child health is vital for a number of reasons. To begin with, economic stability depends on children being able to become the future generations of workers and parents (Wadsworth & Butterworth 2006). Child health is also important because it predicts adult health (Wadsworth & Butterworth 2006, p.31). Additionally, child health is often used as a gauge for national health and economic well-being because

it is a useful proxy for the national standard of living. Child health is particularly important for AI/ANs because children comprise approximately 33% of the total AI/AN population (U.S. Census Bureau 2003 & 2005, see Figure 1.1).[2] The *objective* of this research is to determine the association between child health and selected child characteristics, socioeconomic characteristics, and environmental factors for AI/AN children. The child characteristics included in the analyses are sex, age and birthweight. The SES characteristics are family income, parental education, whether or not both parents are in the home, and the family's health insurance status. The environmental factors include both parental behaviors and parental health. The behaviors include parental drinking (alcohol) and smoking. The health measures include self-rated health, weight status, and whether or not the parent interviewed has asthma. Only one parent and one child per household were interviewed in the data used for this research.

Figure 1.1 Age Distribution for AI/ANs by Age Compared to the U.S. Overall.



[2] Taken from the U.S. Census Bureau International Data Base (IDB) – a computerized data bank containing statistical tables of demographic and socioeconomic data for 227 countries and areas of the world. Last updated April 2005.

1.2 SPECIFIC AIMS

The goal of this dissertation project is to investigate health outcomes that are both salient for the target population (AI/AN children) and available in a nationally representative data set. The specific aims are to:

1. Evaluate the risk/protection of *child characteristics* for selected health outcomes (asthma, ear infections, health limitation, and injury).
2. Determine what *family socioeconomic characteristics* affect selected health outcomes (either positively or negatively) for children living in the same household as their parents.
3. Determine what *environmental (parental) health behavior characteristics* affect selected health outcomes (either positively or negatively) for children living in the same household as their parents.

As this project is focused on AI/ANs, this research will also seek to determine what, if any, characteristics of the child, family socioeconomic characteristics, and/or environmental (parental) behavioral characteristics place AI/ANs at risk for poor health outcomes. In addition, this project will determine whether risks for poor health perform differently across race-ethnic groups. This will advise what risks, if any, are more or less salient for AI/ANs compared to other race-ethnic groups. As no other research has looked at AI/ANs in this regard, this project will add information to current literature.

1.3 RESEARCH DESIGN

There are numerous gaps in the demographic and public health literature for AI/AN children. While studies do exist, few use nationally representative data. The strength of studies at the tribal-level is homogenous samples. The weakness of such studies is that the results cannot be generalized to formulate national policy. It is true AI/ANs are an extremely heterogeneous, including differences in geographic location

(regional, state, urban/rural, and reservation/nonreservation), tribal affiliations, and cultural differences. However, it is arguably more useful to analyze this population as a whole, rather than to ignore them because appropriate, diverse data are unavailable. Barnes et al (2005) echo this sentiment:

> As with other populations, it is likely that there is heterogeneity within the AI/AN population in terms of demographic characteristics as well as health outcomes. Grouping all AI/AN[s]… into a single category may mask the differences among AI/AN subgroups. Other factors that may be associated with health outcomes, including tribal affiliation, [cannot be] examined… due to limitations on sample sizes and available information. Notwithstanding the limitations… the NHIS provides an opportunity to merge data across years and thus increase the precision of the estimates for the AI/AN population. In addition, the data are nationally representative, allowing generalization of these findings to the U.S. population as well as the AI/AN population (p.5).

This research will contribute to filling at least some of the gaps in this literature by using nationally representative survey data (the National Health Interview Survey [NHIS]). This project utilized NHIS data from 1997 to 2003. The health outcomes evaluated in this research are: asthma, ear infections, health limitation, and injury. These outcomes are selected for two reasons: (1) these outcomes are available in the selected data, and (2) these outcomes are salient for the target population. Each outcome will be compared across race-ethnic groups for children between ages 0 and 17. The research will also address three levels of risk/protection for child health outcomes: child characteristics, socioeconomic status, and environmental factors.

1.4 THEORETICAL APPROACH

Healthy People 2010 has two major goals, one of which is to eliminate health disparities (U.S. Department of Health & Human Services 2000). Some health disparities across race-ethnic groups have declined over time, yet continue to persist with negative consequences especially among vulnerable populations (Agency for Healthcare Research and Quality [AHRQ] 2005). Figures 1.3 through 1.6 shown at the end of this section demonstrate this trend using markers developed by AHRQ. Learning about health disparities in children is especially valuable because child health is a major determinant of adult health (Public Health Agency of Canada [PHAC] 2004, Wright et al 2001). The *theoretical approach* for this research is supported by the National Institutes of Health (NIH), which argues health disparities are the result of the complex interaction between biological factors, the environment (social and physical), and specific health behaviors (DHHS 2000).

The role of biology and genetics in health disparities between race groups is currently under debate (Fine, Ibrahim & Thomas 2005, Krieger 2005). However, the role of genotypes in evaluating health is beyond the scope of this project. Biology is approximated in this research using child sex, age, and birthweight as predictors of selected child health outcomes. Birthweight, in particular, has a demonstrated effect on the selected outcomes, save injury (Rovers, de Kok & Schilder 2006, Sin et al 2004, Chen & Millar 1999). Note, however, the context in which these variables were analyzed is one of social science inquiry, not natural science. Thus, these variables will be discussed as social predictors of health, not as biological markers per se. Additionally, parental asthma is used in the analysis of child asthma as a crude measurement of biological inheritance. This project also focused on the other causal factors identified by

NIH, family *environment* and parental *health behaviors* as predictors of selected child health outcomes. All variables were evaluated to determine what risk they may pose to, as well as what protection they may offer against, poor health outcomes. These predictors of the health outcomes were chosen based on extant literature.

A child's environment is, in part, socially determined based on his or her race-ethnicity. Nancy Krieger (2000) uses *ecosocial theory* to explain how researchers can explore the relationship between race-ethnicity and health. This theory seeks to explain how populations express their social experiences biologically through health, disease, and well-being patterns. The causal components of this theory are (1) access to resources (power, property, and means of production) and (2) biology (Krieger 2000, p.39). The role of biology in this research has been explained (above). Most social scientists agree that race-ethnic discrimination limits access to resources (Link & Phelan 2002, 1996, 1995). Unfortunately, it is not possible to directly measure race-ethnic discrimination, or to quantify how such discrimination limits access to resources using the selected data. Instead, access to resources has been approximated using socioeconomic status (SES).

The effect of SES on health has been well established in public health literature (PHAC 2004, Berkman 2000, Lynch & Kaplan 2000, Link & Phelan 2002, 1996, 1995). Higher SES provides protection against poor health and lower SES poses a risk for poor health (Link & Phelan 1995) [see Figures 1.2–1.5]. A recent study on child health in Europe found this to hold true. Children of parents with high education and wealth rated their health-related quality of life as better than those of parents with low education (von Rueden et al 2006). The use of SES in social science has a long tradition, particularly linked to the writings of Karl Marx and Max Weber. These pioneers of sociology articulated social class and how it could be measured empirically. Link and Phelan (2002, 1996, 1995) use a Marxist approach to describe how SES is associated with health

outcomes in their seminal work. People with higher SES have access to resources (i.e., money, knowledge, power, prestige, social support, social networks) that can be used to avoid risks or to minimize the consequences of exposure to risk (Link & Phelan 1995, p.87).

In this research, SES was measured using family income, parental educational attainment, and source of health insurance. Interestingly, another facet of Weber's work is applicable here, as well. The target population, AI/ANs have a "special status" in regard to health insurance because many have access to the Indian Health Service (IHS), a system of affordable health care available to AI/ANs who are members of federally recognized tribes. Thus, even though most AI/ANs do not belong to a high SES group,[3] they do have at least one major resource to help them avoid risk and to mitigate exposure to risk.[4] To fully understand the child's SES context, a variable about home was also included, whether one or both parents live in the household with the child.

To understand the child's environmental context, parental health behaviors such as use of alcohol and parental smoking were measured. There is substantial evidence that anything above a moderate use of alcohol has damaging health effects (NIAAA 2006). Aside from maternal use of alcohol during pregnancy, there is little evidence that exposure to parental drinking of alcohol has a direct effect on child health. There is evidence that exposure to parental drinking of alcohol has an indirect effect on child health, particularly in terms of child injury and behavioral problems (Child Trends Data Bank 2004, Barber & Crisp 1994, Zeitlin 1994, Connolly et al 1993, Bijur et al 1992). This research sought to further explore the indirect relationship between parental drinking and child health. Environmental tobacco smoke has proven to have adverse effects on

[3] According to the National Health Interview Survey (NHIS) weighted data 1997-2003, over 67% of AI/ANs are poor or near poor.
[4] Nearly 40% of the AI/ANs in the NHIS weighted data have IHS listed as a source of insurance.

child health, particularly on respiratory function (Feinson & Chidekel 2006, Hawamdeh, Kasabeh & Ahmad 2003, Tamim et al 2003, Crombie et al 2001, Ehrlich et al 2001).

Several other environmental factors are considered here due to their established influence on child health. They include parental self-rated health, parental weight status, an accumulation of parental health behaviors, and parent asthma. Self-rated health is an excellent predictor of morbidity and mortality in adults (Eriksson, Undén & Elofsson 2001). It is less known though how/if a parent's self-rated health is a predictor of child outcomes. This research attempted to discern whether or not there was an association between these variables. Parental weight status is important, because children whose parents are obese are considered at-risk for obesity themselves (Fowler-Brown & Kahwati 2004, Kanda, Kamiyanna & Kawaguchi 2004, Hood et al 2000, Strauss & Knight 1999, Whitaker et al 1997, Nguyen et al 1996). This offers a unique insight into the child's environment vis à vis family dietary habits (i.e., nutrition and exercise). To fully understand how negative parental health behaviors impact child health outcomes, a variable was created to capture the presence of one to three negative behaviors (drinking alcohol, smoking, and obesity). The operationalization of the variables mentioned here is described in detail in the next chapter.

Figure 1.2 Measures of Quality of Care (AHRQ 2005, p.3).

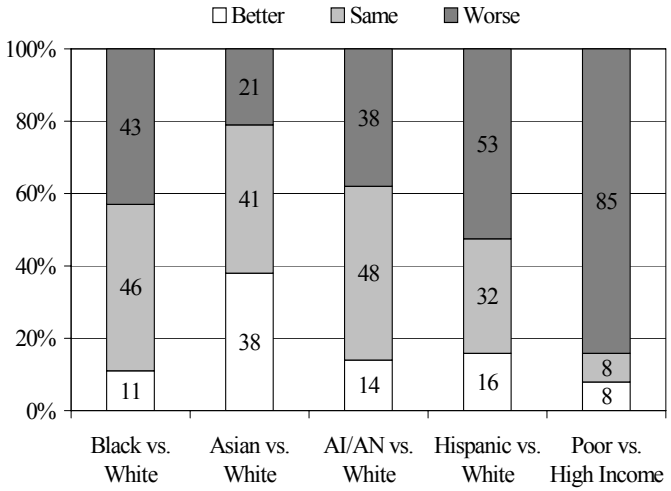


Figure 1.3 Measures of Access to Care (AHRQ 2005, p.4).

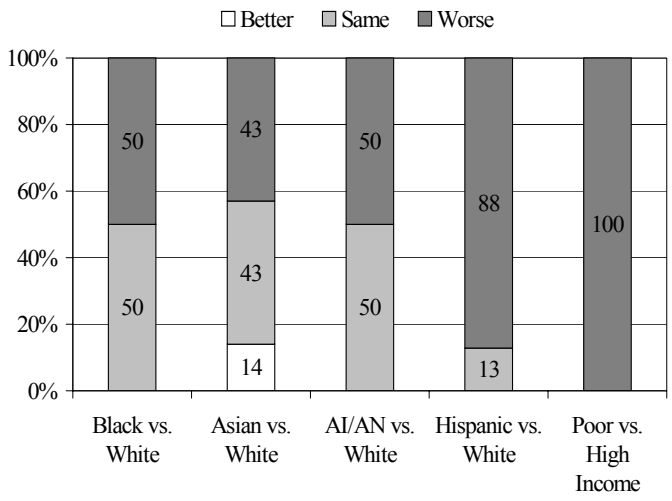


Figure 1.4 Change in Disparities Regarding Quality of Care (AHRQ 2005, p.5).

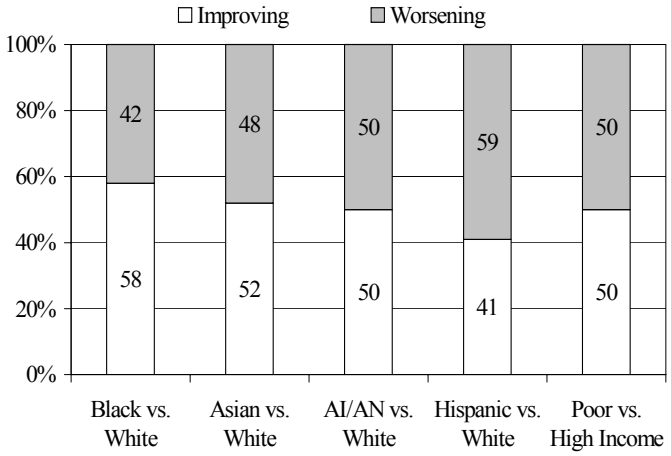
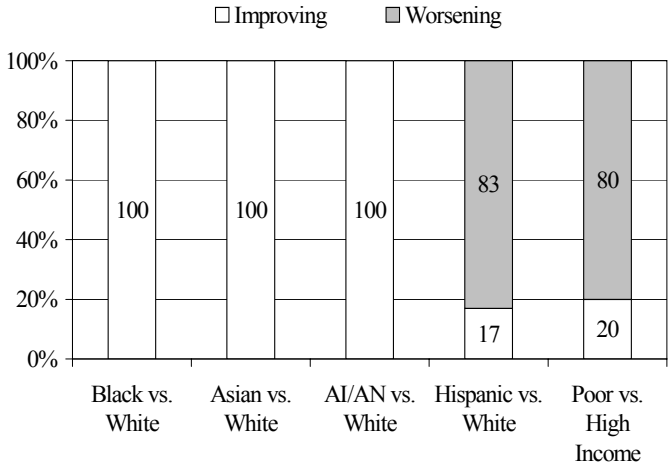


Figure 1.5 Change in Disparities Regarding Access to Care (AHRQ 2005, p.6).



1.5 HYPOTHESES

The results of this research are expected to support those in current literature. This section identifies *hypotheses* for this research. To begin with, disadvantaged minority children are likely to have higher frequencies of asthma (Higgins, Wakefield & Cloutier 2005) and injury (Wallis, Cody & Mickalide 2003) than their non-Hispanic White counterparts. Asian and Mexican origin children are expected to have the same or lower rates of the selected outcomes compared to non-Hispanic Whites. Asian children, while a minority, are not necessarily disadvantaged,[5] and are not expected to have poor health outcomes. Mexican origin children are projected to fare as well as non-Hispanic Whites as implied by the epidemiologic paradox, the public health phenomenon such that persons of Mexican origin exhibit morbidity and mortality outcomes more similar to non-Hispanic Whites than to non-Hispanic Blacks, even though persons of Mexican origin are much more similar to non-Hispanic Blacks than to non-Hispanic Whites socioeconomically (Morales et al 2002, Markides and Coreil 1986). There has been debate over the epidemiological paradox as some researchers believe this phenomenon is a data artifact (Palloni & Morenoff 2001). However, recent findings suggest the paradox is an acurate portrayal of a Mexican Origin health advantage as compared to non-Hispanic Blacks, and not a data artifact (Elo et al 2004). In the case of ear infections, also called otitis media, Blacks and Asians are expected to have fewer diagnoses (Vernacchio et al 2004). Non-Hispanic Whites and Blacks are expected to have reported at least one child health limitation compared to other groups (Child Trends Data Bank 2005b).

A number of additional characteristics are expected to be risk factors for the selected outcomes. In general, boys engage in riskier behavior and are more likely to be

[5] According to the NHIS weighted data 1997-2003, over 66% of Asians have a family income ratio of 2.00 or more to the poverty threshold.

more exposed to hazards than girls, particular risks of injury (Morrongiello & Rennie 1998). Also, there is evidence that boys and girls differ in physical development, placing boys at greater risk for poor health (Schatz, Clark & Camargo 2006, Becklake & Kauffmann 1999). Older children are expected to have higher rates of asthma and health limitations because they have had more time to be exposed to risk. The association between age and injury is not clear. One the one hand, older children have shown a propensity for risky behavior compared to younger children (Morrongiello & Rennie 1998). On the other hand, more children under age four die due to accidental injury than children aged five to fourteen (Wallis, Cody & Mickalide 2003). That said, older children are expected to have lower rates of ear infections, because children usually "outgrow" this type of infection by age five (Vernacchio et al 2004, Freid, Makuc & Rooks 1998). The effect of birthweight on these outcomes is not clear. It is suspected that low birthweight will be a risk factor for asthma (Wjist et al 1998), ear infections (Rovers, de Kok & Schilder 2006), and health limitations (Avchen, Scott & Mason 2001). However, evidence is limited and contradictory. For example, some argue that low birthweight is a risk for reduced pulmonary function (Wjist et al 1998), while others argue it is not (Matthes et al 1995). Still others argue it is high birthweight that presents a risk for asthma (Sin et al 2004). Low SES is also expected to be a risk factor for poor child health (Rovers, de Kok & Schilder 2006). Children whose parents provide a negative health environment (drinking, smoking, poor eating habits) are also expected to have higher rates of poor health outcomes (Child Trends Data Bank 2005a & 2005b, Hood et al 2000, Strauss & Knight 1999).

Finally, there will be characteristics that provide protection against poor child health. Normal birthweight and a high SES should be protective (Rovers, de Kok & Schilder 2006, Sin et al 2004, Avchen, Scott & Mason 2001, Wjist et al 1998, Link &

Phelan 1995). It is not clear if parent's positive health behaviors will be protective against risk for poor child health. Positive behaviors may be protective indirectly to the extent that a child emulates his/her parent's health behavior (Child Trends Data Bank 2006 & 2004, Velleman, Templeton & Copello 2005). However, it is not clear whether, for example, parental *lack* of smoking will act as a protective factor against asthma in the same way parental smoking acts as a risk factor for asthma.

In regards to the target population, it is expected that the following variables will place AI/ANs at risk for poor health: low and very low birthweight, poverty, living in a single parent, and negative parental behaviors. It is also expected that birthweight and negative parental behaviors will be more salient (have a greater impact) on AI/ANs than for other race-ethnic groups. The "special status" of AI/ANs and access to Indian Health Services should mitigate the effects of poverty and living with only one parent. This hypothesis is in accordance with the theory of resource substitution, which suggests having any resource will greatly benefit those with few resource alternatives (Mirowsky & Ross in Hill & Needham 2006). There is little to no literature that informs on the expected results for this hypothesis. In this regard, this research is exploratory.

1.6 ORGANIZATION OF DISSERTATION

This project is presented as a dissertation in five chapters. Chapter one is an overview of the research. Chapter two describes the data and methodology in detail. Chapter three is dedicated to the outcome variable asthma, the only outcome with significant findings for the target population. The remaining outcomes: ear infections, health limitation, and injury are reviewed in chapter four. These outcomes are condensed in a single chapter for two reasons; there was not much literature to review (compared to asthma) and the results were not significant for the target population. Chapters three and four contain brief reviews of salient, extant literature for asthma and the other outcomes,

respectively. These chapters will also provide the respective results as well as a discussion of findings. The final chapter is a summary overview of this project and its implications. It concludes this project and looks toward future research.

Chapter 2, Data and Methods

2.1 DATA SET

The National Health Interview Survey (NHIS) has been conducted annually since 1957. Households are the main unit of collection. Each household is interviewed by trained interviewers who use computer-assisted personal interview (CAPI) forms (interview form is on a laptop). The data are nationally representative of civilian, noninstitutionalized persons. Participants are selected based on the previous decennial census. The structure of the design and collection for the data used in this project is consistent with NHIS procedures dating back to 1985. There are, however, a few changes that bear noting. First, the 1995-2004 design oversampled African American and Hispanic populations. In the previous surveys, only African Americans were oversampled. Second, the number of primary sampling units increased from 198 to 358 between the 1985-1994 design and the 1995-2004 design. Third, due to recent budget cuts, the 2002 household sample was reduced by about 10% compared to previous years.

This research used several of the data files produced by NHIS, 1997-2003. The child sample for each year was merged with the corresponding adult sample to identify the majority of predictive and outcome variables. However, some data were retrieved from the family, person, and injury files, as well as the imputed income files. The "final weight" for the child sample data file was used in the analysis to ensure that the data are nationally representative. Child weight was used because the child is the unit of analysis. The sample consisted of 67,903 children and to child records where the corresponding adult is a non-pregnant parent. Parents in this sample were either biological, adoptive, or step parents. Pregnant mothers were dropped from the analysis because their body mass index did not represent their normal weight.

2.2 DESCRIPTION OF VARIABLES

The variables used in this research are well established in the literature. They have been organized as child characteristics, sociodemographic characteristics, environmental factors, and outcome variables and are listed in Table 2.1. Additional comments are available on each variable as necessary. All of the variable categories in this analysis were dichotomous, coded "1" if the category is applicable and "0" if not.

Several of the variables listed below were rampant with missing values. These values were imputed to maintain the integrity of the sample. As morbidity is a rare event, losing any cases because of missing values would have skewed the results. Missing data were imputed using PROC MI, the multiple imputations procedure, in SAS® 9.1 software. Multiple imputation is superior to single imputation because:

> …Single imputation does not reflect the uncertainty about the predictions of the unknown missing values, and the resulting estimated variances of the parameter estimates will be biased toward zero (Rubin 1989, p.13). Instead of filling in a single value for each missing value, multiple imputation (Rubin 1967; 1987) replaces each missing value with a set of plausible values that represent the uncertainty about the right value to impute. (SAS OnlineDoc™, p.131)

By default, PROC MI creates five imputations of plausible values for each observation's missing data. Imputations are based on 300 Markov Chain Monte Carlo (MCMC) iterations, which is a general approach for simulating conditional distributions in statistics. The first 200 MCMC iterations (called "burn-ins") are discarded because they can produce imputed values that are highly dependent on the previously imputed values as well as on the values used to start the simulation. The last 100 MCMC iterations can be regarded as a random sample from the (stationary) joint posterior

distribution. The posterior distribution can be viewed as the distribution of missing data values and any other unknown parameters of interest. Sample means calculated from this distribution are the imputed missing data values. The MCMC method is appropriate when the pattern of missing data is arbitrary as opposed to monotonic (Schafer 1997, as cited in SAS OnlineDoc™).

The EM algorithm is used to obtain starting values for the MCMC iterations (Dempster, Laird, Rubin 1977). The EM algorithm uses the observed data to estimate the posterior means and (co)variances of the distribution of the missing data. Uncertainty about the means and (co)variances of the missing data is handled by assigning a noninformative prior distribution to these parameters using a full probability model. Specifically, the data are assumed to be multivariate normal and the parameters underlying the data are treated as random variables with prior distributions.

From this (Bayesian) perspective, the missing data have a distribution that is identical the probability distribution of the corresponding observed data. Imputation simulates missing data for each observation independently, conditional on the observed data for that observation using MCMC. Specifically, the MCMC iterations generate a posterior predictive distribution of complete data (observed data + missing data) based on sample data and prior information. Sampling from this distribution provides data values which are averaged to obtain point estimates of missing data values for a particular observation. Repeated sampling from the posterior predictive distribution provides alternative estimates of missing data (i.e., the multiple imputations) for a particular observation.

PROC MI also requires a seed to generate a random number. The website www.random.org was used to produce a separate seed for each procedure. The mean of the five imputations was calculated separately and used to replace the missing value. The

mean for the imputed data was compared to the mean of the original data to ensure there were no large differences. Table 2.1 also demonstrates the distribution of this sample for each variable. This distribution is post-imputation. Distributions by child's race-ethnicity are shown in Appendix A.

2.2.1 Child Characteristics

The characteristics of the child included both exogenous and predictor variables. The exogenous variables were race-ethnicity, sex, and age. The predictor variable was birthweight.

Race-ethnicity

Race-ethnicity was taken from the single race category in each data file and was coded as: non-Hispanic White, non-Hispanic AI/AN, non-Hispanic Black, Mexican Origin, non-Hispanic Asian, non-Hispanic Other or Multiple Race, and Other Hispanic. There were .05% cases missing for this variable. Missing data for this variable include cases where race could not be reported due to confidentiality reasons (NHIS Data Documentation 1997-2003). Note that for years 1997 and 1998, non-Hispanic Asian will include Pacific Islanders. The race category for Asians and Pacific Islanders was inseparable for these two years. Also note the category "other race" was not available in 2003. Missing data for this variable were imputed in two stages. First, missing cases were set to match parent's race. If parent's race was not available, then missing cases were imputed using the PROC MI.

It should be noted that the variable that identifies AI/AN is self-reported. Rather, the responding adult identified the selected child as AI/AN. No authentication is required to self-identify as an AI/AN in the NHIS data, nor on the Census, or other self-report data. Further, American Indians and Alaska Natives are collapsed into a single race-ethnic category and cannot be separated. Therefore, it is not possible to discern if the

children reported as AI/AN here are part of a tribe, a federally recognized tribe, live on a reservation, or receive any benefits associated with being an AI/AN.

It is widely believed that many who self-identify as AI/AN are not "officially" AI/ANs. Between 1960 and 1990, the number of Americans reporting American Indian as their race in the U.S. Census more than tripled, growing from 552,000 to 1,959,000 (Passel 1997). Even though AI/ANs have more children than non-Hispanic Whites (Snipp 1997), this population increase was not due to fertility nor immigration (Passel 1997). Instead, this increase was a result of an increase in self-identification as American Indian (Passel 1997). It is not known whether there was a similar increase in self-identification as Alaska Native.

Age

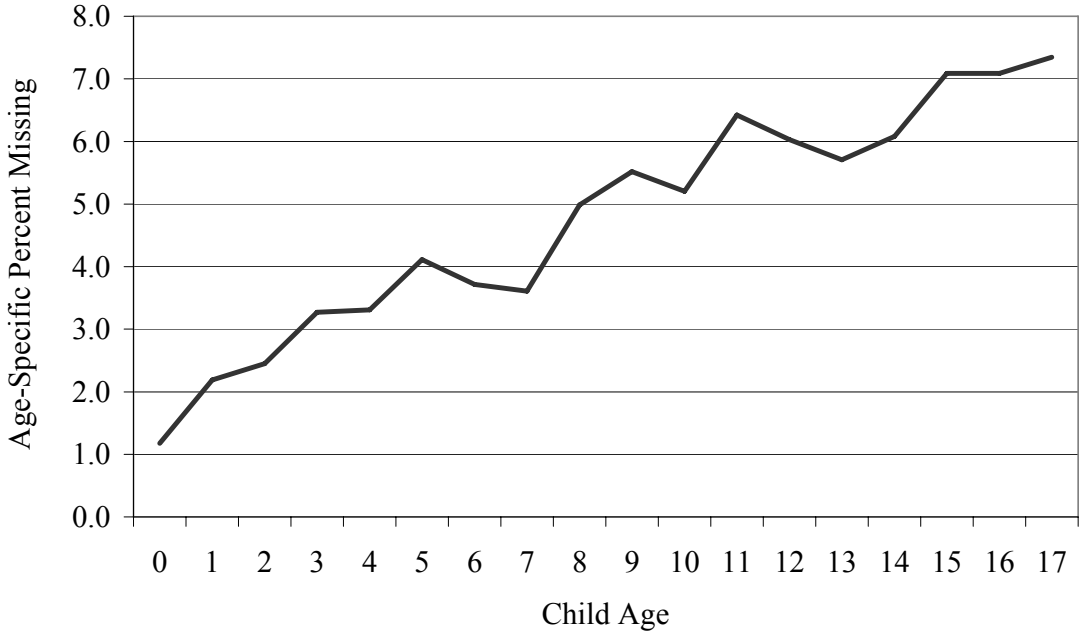
Child age was measured in cut-points. In the race-inclusive models, age was divided into four categories: 0-3, 4-7, 8-11, and 12-17. In the race-specific models, age is collapsed to accommodate smaller Ns: 0-4, 5-11, and 12-17. All ages available in the NHIS data were used to maximize the number of children included in the analysis. It important to note, however, that using all ages introduced heterogeneity into the analysis. This is especially true in the case of child injury. Injuries sustained by very young children (infants) are usually the results of actions taken (improper handling or abuse) or not taken by the supervising adult (lack of supervision). As children age, they gain autonomy in the behavior and, thus, contribute to their own injuries, whether the injuries be sustained through extracurricular activities or narcotic abuse.

Birthweight

Researchers have validated the accuracy of mother's recall of birthweight (Olson et al 1997, Seidman et al 1987). In all, 4.7% of the cases were missing birthweight in this NHIS sample. Both mothers and fathers were respondents, but missing birthweight did

not appear to be correlated with parent-status of respondent. Of the cases with missing

Figure 2.1 Percent Distribution of Missing Birthweight by Age, NHIS 1997-2003 Unweighted.



birthweight, 50.8% had mother respondents and 49.2% had father respondents. Instead, it appears missing birthweight data was a recall issue correlated with child's age. The older the child, the more likely the parent was to forget the child's birthweight (See Figure 2.1).

Table 2.1 Variable Definitions and Weighted Percent Distributions.

Variable Categories	Percent Distribution
CHILD CHARACTERISTICS	
Race	
Non-Hispanic White	66.27
Non-Hispanic AI/AN	0.70
Non-Hispanic Black	13.10
Mexican Origin	9.64
Non-Hispanic Asian	3.02
Non-Hispanic Other	1.76
Other Hispanic	5.50
Sex	
Male	51.18
Female	48.82
Age	
0-3	21.55
4-7	22.48
8-11	23.57
12-17	32.41
Birthweight (grams)	
Very low (< 1500)	1.05
Low (1500-2499)	5.59
Normal (2500-3999)	80.11
High (4000 +)	13.25
SOCIODEMOGRAPHIC CHARACTERISTICS	
Family Income to Poverty Ratio	
Poor (< 1.00)	17.28
Near Poor (1.00-1.99)	20.40
Not Poor (> 2.00)	62.32
Parental Educational Attainment	
No high school diploma	16.02
High school diploma or GED	28.22
Some college	30.50
College degree	25.26
Parent Household Composition	
One parent in household	23.63
Both parents in household	76.37
Insurance Status	
No insurance	7.01
Government insurance	17.68
Private insurance	75.31

ENVIRONMENTAL FACTORS	
Parent Use of Alcohol	
Does not drink	34.53
Light drinker	57.01
Moderate or heavy drinker	8.45
Parent Smoking Status	
Never smoked	58.80
Former smoker	16.91
Light smoker	6.57
Heavy smoker	14.38
Heavy plus smoker	3.34
Parent Self-rated Health	
Excellent or very good	71.75
Good	21.54
Fair or poor	6.71
Parent Weight Status (BMI)	
Underweight (< 1850)	1.85
Normal (1850-2499)	39.01
Overweight (2500-2999)	33.39
Obese (3000-3999)	19.40
Morbidly obese (4000 +)	2.58
Weight unknown	3.77
Accumulation of Parent Health Behaviors	
No negative behaviors	27.86
One negative behavior	54.26
Two negative behaviors	16.03
Three negative behaviors	1.85
Genetic Proxy for Asthma	
Parent has asthma	9.72
Parent does not have asthma	90.28
Outcome Variables	
Asthma	11.96
3+ ear infections	6.73
Health limitation	2.12
Injury	2.74

2.2.2 Sociodemographic Characteristics

These variables were designed to capture financial and home stability. The variables that speak to financial security include family income, parent educational status (as a representation of potential income and the ability to access and utilize resources [Mirowsky & Ross 2003]), and health insurance status. The other variable associated with home stability was parental household composition.

Ratio of Family Income to Poverty Threshold

This variable is available on the NHIS family data file. Family income was calculated by combining the incomes of those persons in the same family, in the same household. The ratio to the poverty threshold ranged from under .50 to 5.00 and above. Poverty was defined as a ratio below 1.00. Near poverty was defined as a ratio of 1.00 to 1.99. Not poor was defined as a ratio of 2.00 and above. The data available of the family file included many missing cases. Fortunately, NHIS addressed the problem and provided "Imputed Income Files" for each of the years in this analysis. These imputed values were incorporated into the final data for this project.

Parental Educational Attainment

All variables that refer to parents include biological, step and adoptive parents. Parent's exact relationship to the child was not available in all data years. Parental education was located in the adult file and is measured by the question, "What is the HIGHEST level of school [you have] completed or the highest degree [you have] received?" This is a useful measurement because education is not simply a measurement of years in school, but also provides certification achievements. Missing data (0.61% of weighted analysis data) for this variable were imputed using PROC MI in SAS software.

Parent Household Composition

Parents' marital status does not always determine whether both parents are in the household. This variable was available on the family file and tells whether the child lives with one or both parents. There were no missing cases in the analysis data for this variable.

Source of Insurance

This variable was available on the family data file. It is not possible to tell if the child is actually covered by insurance based on this variable. At best, it can gauge whether anyone in the family has insurance, including private insurance (coded as "private insurance"); Medicare, Medicaid, military coverage, state-sponsored health care, Indian Health Service, or other government-sponsored insurance (coded as "government insurance"). There were no missing cases in the analysis data for this variable. In the AI/AN race-specific models, the variable "no insurance" was replaced with "Indian Health Services." This was necessary for two reasons: (1) there were only 12 cases of AI/ANs with no insurance, an incidence rate far below the other race-ethnic groups, and (2) Indian Health Services is a unique and pervasive health care system available only to AI/ANs. It did not make sense to include this variable in other race-specific models, nor to exclude it from the AI/AN models.

2.2.3 Environmental Factors

These factors are designed to capture the child's exposure to certain parental behaviors. While these behaviors are likely to occur in the home, they are not limited to the home environment. In other words, if a parent smokes heavily, it follows the parent smokes in and/or around the home, and possibly in the car, at social functions, and anywhere else the parent and child might spend time together.

Parental Use of Alcohol

Heavy drinking has been defined as drinking alcohol two to seven days a week and, on the average, having at least five drinks on the drinking day (OAS 2001, Presley et al 1996). More recently, Fillmore et al (1998) advocate using three indicators of alcohol consumption: quantity, frequency, and volume. Quantity (Q) is the number of drinking occasions per month, frequency (F) is the average number of drinks per month, and volume (V) is the product of quantity and frequency (V=Q*F). Volume is used in the research to determine parental drinking status.

NHIS data were unable to capture number of drinks per sitting because of the wording of the questions. Instead, the best estimate of this standard was to gauge number of drinks per drinking day, rather than drinking occasion. This is derived from combining responses to two questions on the adult survey, "In the past year, how often did you drink any type of alcoholic beverage?" and "On those days that you drank, on the average, how many drinks did you have?" Those who drink but are not heavy drinkers are identified as light drinkers. Light drinking is considered safe by the Harvard School of Public Health (2004). It also considered by many to be a healthy behavior (Auger et al 2002, Criqui & Ringel 1994, Klatsky, Armstrong & Freidman 1990, and Mikhailidis et al 1986). This variable will also identify those who did not drink in the past twelve months.

Fillmore et al (1998) are careful to point out identifying levels of alcohol consumption should vary by sex. Following their guidelines, the following parameters are used:

Lifetime abstainer	=	< 12 drinks in lifetime
Former drinker	=	12+ drinks in lifetime and none in the past year
Light drinker	=	Women: V < 21 Men: V < 30
Moderate drinker	=	Women: 21 <= V < 30 Men: 30 <= V < 43
Heavy drinker	=	Women: V >= 30 Men: V >= 43

NHIS also allows for identifying those who are frequent heavy drinkers in the question, "In the past year, on how many days did you have five or more drinks of any alcoholic beverage?" This may prove to be a useful distinction considering the high rates of alcoholism on Indian reservations (Ehlers & Wihelmsen 2005, Beals et al 2003, NIAAA 2002, and Beauvais 1998). Drinking status "unknown" includes people who had 12+ drinks in lifetime, but refused to reply or did not ascertain how often (represented by codes: 997 = refused, 998 = not ascertained, 999 = unknown). They were not asked about the amount. Missing cases (2.44% of the weighted analysis data) were imputed.

Parental Smoking

A meta-analyses of data on smoking concluded that self-reports of smoking are accurate in most studies (Patrick et al 1994). Heavy smoking is defined as having at least 15 cigarettes a day (CDC 1998). The Department of Health and Human Services defines heavy smoking as "smoking an average of a pack or more of cigarettes a day in the past month (OAS 1999)." The NHIS adult data survey contains a question on cigarettes smoked per day. A pack of cigarettes can vary from 10 to 25 cigarettes per pack. Light smokers are those who smoke but are not heavy smokers. This variable was used to

identify parents who do not smoke (current non-smokers are not in the universe for the question).

Respondents who answered "some days" or "everyday" to "Do you NOW smoke cigarettes every day, some days or not at all?" were selected for identification of smoking quantity. Quantity was measured by the response to "On the average, how many cigarettes do you now smoke a day?" Light smokers were defined as smoking less than 10 cigarettes a day. Heavy smokers are those who smoke between 10 and 24 cigarettes a day. Heavy plus smokers are those who smoke 25 or more cigarettes a day. Former smokers were identified as those who responded to the question "How long has it been since you quit smoking cigarettes?" Those who answered "no" to "Have you smoked at least 100 cigarettes in your ENTIRE LIFE?" were coded as "never smoked."

There were missing cases for this variable. 1.04% of the weighted sample responded "some days" or "everyday" to the question "Do you NOW smoke cigarettes every day, some days or not at all?", then failed to report a number of cigarettes per day. Missing cases (1.04% of the weighted analysis data) were imputed.

Self-Rated Parent Health

Self-rated health is a robust measure of health status (Subramanian & Kawachi 2006, Desalvo et al 2005, and Weich, Lewis & Jenkins 2002). The question associated with this variable is "Would you say [your] health in general is excellent, very good, good, fair, or poor?" Missing cases (.06% of the weighted analysis data) were imputed.

Parental Body Mass Index

Parents' body mass index (BMI) was tabulated using self-reported height and weight from parents in the adult sample. Note that because parental BMI was being used as an environmental mediator, it was not limited to biological parents. In the adult sample "BMI was calculated using the inhouse version of the height and weight

variables, which contain the greater range of height and weight values than are available on the public use file. BMI = [Weight (kg)/[Height(m) squared]] rounded to 2 decimal places (NHIS 2003)." The Surgeon General recommends using the following cut-points to ascertain weight status for adults (DHHS 2003c, CDC). The category of "morbidly obese" was added to test for variation. These cut-points are generally accepted and utilized in most health research:

BMI Range	Status
0 – 18.5	Underweight
18.5 – 24.9	Normal
25.0 – 29.9	Overweight
30.0 – 39.9	Obese
40.0 +	Morbidly Obese

Body mass index is commonly used as a measure of health status and is a measure of body fat. NHIS uses this measure because other methods require clinical training and personal contact, such as the skin-fold method. Body mass index, though widely used, does have several limitations. For example, BMI does not consider percentage body fat or body frame (Baumgartner 2000). Those adults who have a high BMI due to muscle mass may be categorized as obese. Another limitation of this variable is due to heaping. Women tend to self-report weight lower than true weight and men tend to self-report height taller than true height (Rogers, Hummer and Nam 2000). The National Center for Health Statistics (NCHS) discourages imputing missing values for BMI (personal communication with P. Meyer at NCHS), so an additional category of "unknown" is kept in the data.

Accumulation of Parent Health Behaviors

Children's home environment is not limited to one particular parental behavior. This variable captures the accumulation of three parental health behaviors that can have a potentially deleterious effect on child health. Those behaviors are parent use of alcohol,

parent smoking, and parent body moss. The parent behaviors considered "negative" are: moderate or heavy drinking; current smoking (light, heavy, and heavy plus); and body mass of 25.0 or more (overweight, obese, and morbidly obese). The behaviors are selected because they are related to personal locus of control, at some level.

Parental Asthma

This variable is only applicable to the models for asthma. There are no similarly relevant variables for ear infections, health limitation(s), or injury. This variable was a proxy because it potentially represents the child's exposure to asthma beyond social context. If the parent has asthma, this may indicate a genetic predisposition to the condition. This variable is not, however, without limitations. As mentioned earlier, parents in the analysis sample may be biological, adoptive, or step parents. Exact relationships cannot be determined for all years.

In the years 1998 – 2003 relationships to mothers and fathers living in the household were available. The following percent averages were present in the NHIS child samples used in this research, 1998 – 2003 (weighted):

	Biological	Adoptive	Step	Other	Unknown
Mother	96.04	1.53	1.27	0.49	0.68
Father	89.34	1.97	7.45	0.53	0.71

In the weighted analysis sample, 61.7% of the parent-child relationships were mother-child, and 38.3% were father-child. The cases where the relationship is father-child are at greater risk of being non-biological than the cases where the relationship is mother-child.

2.2.4 Health Outcomes

All of the outcome variables were coded as dichotomous variables, as "1" if the outcome occurs, and "0" if the outcome does not occur. Most of the variables were found on the child data file, including asthma, ear infections, and health limitation. The data for

injuries is located on a separate date file for injury and poisoning. The questions used to measure these outcomes in the NHIS are as follows:

Asthma	"Has a doctor or health professional ever told you that [child] had asthma?"[6]
Ear infection	"During the past 12 months, has [child] had three or more ear infections?"
Health limitation(s)	"Does [child] have an impairment or health problem that limits his/her ability to crawl, walk, run, or play?"
Injury	"During the past three months, did you [or anyone in your family] have an injury where any part of [your/the] body was hurt?"[7]

Note that asthma is the only outcome with requisite professional consultation. The question that captures the presence or absence of asthma in the selected child asked if it was diagnosed by a doctor or health professional. This may well introduce a selectivity bias into the analysis in that children who have not been to a doctor or health professional will not have been diagnosed with asthma. As demonstrated later in this text, children in families with low SES measures disproportionately defer doctor visits for more than a year (see Table 3.2). This selectivity bias should not present a problem for AI/ANs, however, as 40% of AI/ANs claimed access to IHS.

The variables health limitation and injury are best understood with additional information. The variable for health limitation is further explained in the NHIS data documentation using the keywords: physical impairment, mobility limitation, and mobility impairment. Examples of health limitations include cerebral palsy, autism,

---

[6] According to Akinbami, Rhodes & Lara (2005), the estimates reported in NHIS data for asthma by race-ethnicity are robust. They found no evidence that symptomatic minority children are underdiagnosed with asthma compared to non-Hispanic White children. However, asthma may be underreported for all children, across race-ethnicity (Edwards et al 1994).

[7] The Injury File includes only those injuries that were *medically attended* (NHIS 2004, p.39). This attention may be minimal, such as a phone call (personal communication with S.J. Jack (at the CDC), March 2, 2006). NHIS advises the injury data file be used with caution because of under-reporting (NHIS 2004, p.39).

hearing impairment and blindness. Specific health limitations are not measurable using NHIS data. The Injury File also provides information on location (place) and cause of injury. The causes of injuries included are transportation, burning/scalding, falling, struck by object or person, cut/pierce, machinery, and other. The injuries included in this research are the first injuries listed for each child. The NHIS files contain data on up to 10 injuries per person. It would be cumbersome to review results for all injuries reported for each child in this report. In addition, the percentage of children with any type of injury is small.

2.3 DATA ANALYSIS

The first step in any analysis was to create numerous crosstabulations to map the relationships between my exogenous variables, predictor variables and the outcome variables. These crosstabulations produced proportions that were compared across race-ethnic groups.[8] Different models were estimated to provide understanding of the nature of race-ethnic differences in asthma, ear infections, health limitations, and injury. Models that included race-ethnicity as an exogenous variable were used to determine what, if any, characteristics of the child, family socioeconomic characteristics, and/or environmental (parental) behavioral characteristics place AI/ANs at risk for poor health outcomes. Models that were race-specific advised whether these variables affect different races in different ways. These models advised what risks, if any, are more or less salient for AI/ANs compared to other race-ethnic groups. Unfortunately, these models did not yield significant results for AI/AN, rendering cross-race comparisons and a meaningful dialogue regarding the target population impossible. Only two of the four

8 The crosstabulations for this paper were produced using SAS software, Version 9.1 of the SAS System for Windows, version 5.1.26. Copyright © 2002-2003 SAS Institute Inc. SAS and all other SAS Institute Inc. product or service names are registered trademarks or trademarks of SAS Institute Inc., Cary, NC, USA.

AI/AN race-specific models contained any significant results at the $p \leq .10$ (or less) level. These findings are reported in the appropriate chapters.

This research used multivariate analysis because it estimated the effect of a predictor variable (child characteristics, SES, and environment) on the dependent variable (health outcome), while holding constant the effects of other, possibly related variables. Logistic regression is useful because it predicts a binary outcome (positive health outcome vs. negative health outcome). Logit modeling is also useful because it offers a convenient interpretation of predictor variables' effects on the log odds of the positive response. The variables in this analysis were all dichotomous, allowing a convenient interpretation of the logistic regression coefficients as odds ratios. Odds ratios are valuable because they demonstrate how much higher or lower the odds are of a positive outcome for a comparison group relative to the reference group. The logit model is preferred in epidemiology, demography, and public health research because of the close analogy between odds ratio and relative risk (Powers & Xie 2000).

In this analysis, the pseudo-maximum likelihood (ML) statistics were treated as if they were ML; then the usual likelihood ratio tests are performed using these statistics. The estimation procedure used by SUDAAN is not precisely maximum likelihood (as is the case for other complex survey software). For each model estimated, a pseudo R^2 statistic was calculated to evaluate model improvement.[9] SAS-Callable SUDAAN does not calculate this statistic automatically; it was calculated separately using the formula:

$$\text{McFadden's } R^2 = 1 - \left(\frac{\ln L_{\text{(Full Model)}}}{\ln L_{\text{(Intercept Only)}}} \right)$$

[9] The Hosmer and Lemeshow Goodness of Fit statistic is often used to report model efficiency (level of discrimination) for logit regression. We do not use this statistic in this analysis because our data, NHDS, is complex survey data, not a simple random sample (Korn, Graubard & Midthune 1997).

The sampling design for NHIS is cluster sampling, which is best analyzed using SUDAAN® 9.01 to estimate standard errors. SAS-callable SUDAAN software (Research Triangle Institute, Research Triangle Park) was used for the analysis. SUDAAN is one of several software packages that can produce standard errors of estimates corrected for design effects (such as the cluster sampling used in the NHIS data). Such alternate programs include STATA (STATA Corp, College Station) and R (R Foundation for Statistical Computing, Vienna). SAS-callable SUDAAN was utilized for this project because of familiarity with SAS (versus STATA or R).

It is important to note the race-specific and race-inclusive models do not utilize identical procedures in SUDAAN. The sampling frame for the NHIS necessitates less precision in the race-specific models, compared to the race-inclusive models. Figure 2.2 illustrates the NHIS sampling frame. The race-inclusive models are nested at both the stratum and the primary sampling unit (PSU) levels. This technique works well for the entire sample (N=67,903 children). The race-specific models are nested only at the PSU level. In these models, N ranges from 371 to 37,789, yielding cell sizes that cannot tolerate precision beyond the PSU. Following are programming examples of the differences between these two modeling procedures:

Race-Inclusive Models

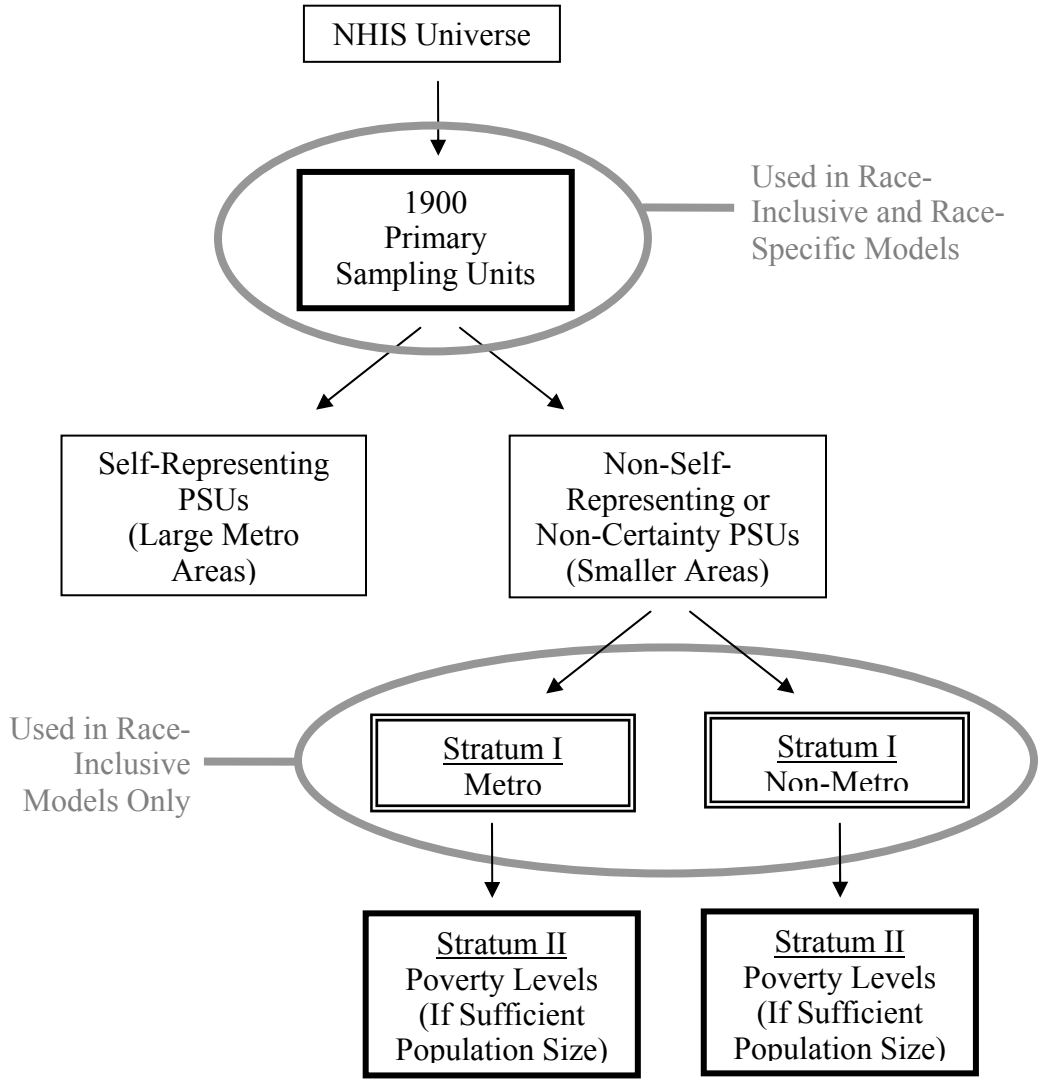
```
proc rlogist design=wr ;
nest stratum psu ;
model asthma = [list predictor variables]
weight WTFA_SC ;
run ;
```

Race-Specific Models

```
proc rlogist design=wr ;
nest psu ;
model asthma = [list predictor variables]
weight WTFA_SC ;
run ;
```

These approaches are also demonstrated in Figure X, using blue font. Using these two different procedures does not affect the analysis substantively. Indeed, the race-specific models do not run when “stratum” is included in the model.

Figure 2.2 Sampling Frame for NHIS Data Collection, 1997-2003.



The findings reported in Chapters 3 and 4 were significant at the $P < 0.1$ level. Altering the significance level from the norm (.05) to 0.1 increased the chance of a Type I error, but lowered the chance of a Type II error. This was an important consideration because AI/ANs were a small proportion in the sample. Thus, the risk of reporting a false negative for the target population was deflated. The race-inclusive regressions were performed in the following order:

Baseline	Race-ethnicity
Model 2	*Adding* Child Characteristics:
	Sex
	Age
	Birthweight
Full Model	*Adding* Family Socioeconomic Characteristics:
(Model 3 for asthma)	Family Income to Poverty Ratio
	Parent Education
	Parent Household Composition
	Source of Insurance
	Adding Parental Health Behaviors:
	Parent Use of Alcohol
	Parental Smoking
	Parent Self-Rated Health
	Parental Weight
(Full Model for asthma)	*Adding* Parental Asthma
Alternate Full Model	*Adding* Accumulation of Parental Health Behaviors
	Removing Parental Health Behaviors (as separate variables)

Many of the variable categories were collapsed/recoded compared to the race-inclusive models to adjust for smaller sample sizes: age, birthweight, poverty status, parental education, parental drinking, parental smoking, parental weight, and accumulation of negative parental health behaviors. Parental health behaviors that were not significant in the race-inclusive models were omitted from the race-specific models. The race-specific regressions were performed in the following order:

Baseline	Child Characteristics:
	Sex
	Age
	Birthweight
Full Model	*Adding* Family Socioeconomic Characteristics:
(Model 3 for asthma)	Family Income to Poverty Ratio
	Parent Education
	Parent Household Composition
	Source of Insurance
	Adding Parental Health Behaviors:
	Parent Use of Alcohol *(in injury model only)*
	Parental Smoking *(not in injury)*
	Parent Self-Rated Health
	Parental Weight
(Full Model for asthma)	*Adding* Parental Asthma
Alternate Full Model	*Adding* Accumulation of Parental Health Behaviors
	Removing Parental Health Behaviors (as separate variables)

Chapter 3, Child Asthma

Asthma is the leading cause of chronic illness in childhood (Grupp-Phelan, Lozano & Fishman 2001). Asthma has been identified as one of the most striking, persistent race-ethnic health disparities (DHHS 2000). Low-income populations, minorities, and children living in inner cities experience disproportionately higher morbidity and mortality due to asthma than middle-, high-income non-Hispanic Whites (CDC Nd, 10/09/06, Higgins, Wakefield & Cloutier 2005, Turyk et al 2006). According the U.S. National Institutes of Health Guidelines for Management and Diagnoses of Asthma, asthma is defined as:

> a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role, in particular, mast cells, eosinophils, T lymphocytes, macrophages, neutrophils, and epithelial cells. In susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment. The inflammation also causes an associated increase in the existing bronchial hyperresponsiveness to a variety of stimuli (p.8).

The Centers for Disease Control and Prevention (CDC) estimates that asthma prevalence among children has been increasing by an average of 4.3% per year in the U.S. (CDC Nd, 10/09/06). The CDC also identifies several reasons why asthma in children warrants public health attention: (1) asthma accounts for 14 million lost days of school missed annually, (2) asthma is the third-ranking cause of hospitalization among those younger than 15 years of age, and (3) the number of children dying from asthma

increased almost threefold from 93 in 1979 to 266 in 1996 (CDC Nd, 10/09/06). This chapter will briefly review current literature as it relates to the variables used to predict child asthma in this research. It will also report results of the logistic regression analysis, as well as offering a discussion of these results.

3.1 LITERATURE REVIEW

This section is organized to review literature on the predictor variables included in the regression models. There are other predictive factors associated with child asthma that cannot be measured in this study, such as immunoglobin (IgE) levels (Nelson et al 1997), the Hygiene Hypothesis (Liu & Murphy 2003), environmental exposure to allergens (Findley et al 2003), and physiological differences (Ramsey et al 2005, Celedón et al 2004). This review, however, only reflects literature salient to this research.

3.1.1 CHARACTERISTICS OF THE CHILD

This section will review the following variables: race-ethnicity, sex, age, and birthweight. There is an established literature arguing that each of these variables is associated with child asthma.

Race-ethnicity

To begin with, there is a large body of work dedicated to explaining the relationship between race-ethnicity and asthma. Much of this work has been site-specific and has found that Black and Hispanic children are at higher risk for asthma than non-Hispanic White children independent of SES (Higgins, Wakefield & Cloutier 2005, Rodriguez et al 2002, Nelson et al 1997). For example, in a study of Southfield, Michigan middle-class families, Nelson et al (1997) found that third grade Black students experienced an asthma prevalence twice as high their Whites counterparts. Puerto Ricans as Hispanics are at risk for asthma (Lara et al 2006, Ramsey et al 2005). There is conflicting evidence as to the risk for asthma among Mexican origin children. One study

conducted in the Texas panhandle provided evidence these children are at lower risk for asthma (OR = 0.48) compared to non-Hispanic Whites (Arif et al 2004). On the contrary, a study using the National Health and Nutrition Examination Survey (NHANES III) data found that Mexican origin children have an OR of 1.20 for asthma compared to Whites (Rodriguez et al 2002). Simon et al (2003) found in Los Angeles that Asian children have the same or slightly less risk for asthma than White children (OR = 0.90). There is some evidence that increased risk for asthma by race-ethnicity occurs only in extreme poverty (Smith et al 2005).

No studies have been conducted to provide national prevalence rates for AI/AN child asthma (Lewis et al 2004, Gessner 2003). Recent estimates suggest that between 6.9% and 9.9% of AI/AN children have been diagnosed with asthma (DHHS 2006, Gessner 2003). These rates are below the national average and below those of other race-ethnic groups. However, there is evidence from Washington State that AI/AN children require higher rates of hospitalization due to asthma than other children (Liu et al 2000). While there is sufficient evidence that asthma is an important health outcome for AI/AN children, no studies were found that compared children from this group to children in other race-ethnic groups.

Sex

Most empirical evidence suggests that boys are at higher risk for asthma than girls (DHHS 2006, Nicholas et al 2005, Findley et al 2003, Nelson et al 1997, Schwartz et al 1990). Using the NHANES II, Schwartz et al (1990) found boys had an OR of 1.4 compared to girls. In a study of child asthma in Harlem, Findley et al (2003) found similar results – boys had an OR of 1.48 compared to girls. Nelson et al (1997) reported the lifetime prevalence for boys in their sample was 14% compared to 5% in girls. Additionally, DHHS (2006) reported 14.8% of boys had been told by a health

professional they had asthma, compared to 9.4% of girls (p.33). However, there are reports that girls are at greater risk for asthma (Lwebuga-Mukasa, Oyana & Wydro 2004, Boardman, Finch & Hummer 2001). For example, Boardman, Finch & Hummer (2001) found that girls were at greater risk for respiratory problems compared to boys (OR = 1.43).

Age

There is some evidence to suggest that older children are at greater risk for asthma than younger children (Lwebuga-Mukasa, Oyana & Wydro 2004, Rodriguez et al 2002). However, there is no study that places emphasis on age as a predictor of asthma. In a study of children in Buffalo, NY, children aged 6-17 had an OR of 3.3 for asthma compared to younger children (Lwebuga-Mukasa, Oyana & Wydro 2004). Additionally, DHHS (2006) reported that a higher proportion of children aged 12-17 were told by a health professional they had asthma compared to younger children.

Birthweight

There is an established literature providing evidence that poor birth outcomes have a negative affect on health outcomes (Ment et al 2003, Wise 2003, Bhutta et al 2002, Boardman et al 2002, Strauss 2000b, McCormick et a1 1992). Public health researchers have coined the terms "programming hypothesis" and "fetal origin hypothesis" to describe the effect birth outcomes have on health through the life course (Rona et al 2005, Edwards et al 2003, Lucas, Fewtrell & Cole 1999). Birthweight is a commonly used measure of birth outcomes and is used in this research to predict asthma. The role of birthweight in the development of asthma is currently under debate. Some researchers believe low birthweight presents a greater risk for asthma, while others argue high birthweight presents a larger risk. Still, others argue that it is not birthweight per se, but gestational age that has an effect on the development of asthma and health limitations

(Raby et al 2004, Foulder-Hughes & Cooke 2003, Woods et al 2003, Falk et al 1997). This research, however, cannot measure gestational age using the selected data.

Using the Aberdeen Maternity and Neonatal Data Bank, Edwards et al (2003) found a positive linear trend between low birthweight and lower adult lung function, controlling for maternal and adult factors (such as maternal and current smoking). Specifically, these authors note subjects born at birthweights in the top four quintiles had better respiratory function that those born at birthweights in the lowest 20%. Consistent with the "fetal origins hypothesis," the authors suggest low birthweight is a risk for poor respiratory function because "retarded weight gain of the fetus…can constrain the growth of airways with effects that persist into late adulthood and old age (Edwards et al 2003, p.1061)." While this study is not of children, it demonstrates a statistically significant relationship between low birthweight and lung function. Boardman, Finch & Hummer (2001) echo this finding in their study of children aged three to four from the National Maternal and Infant health Survey. They found that very low (500–1499 grams) and low (1500–2499 grams) birthweight presented an OR of 3.98 compared to children born at birthweights above 3500 grams. Jaakkola & Gissler (2004) also found that low birthweight (less than 2500 grams) presented a risk for asthma in seven year olds in Finland (OR = 1.69 compared to *not* low birthweight).

On the contrary, Sin et al (2004) suggest the high birthweight presents a risk for childhood asthma severity. They explain the relationship between high birthweight and asthma is mediated and/or moderated by adiposity (obesity). "Adiposity adversely affects lung function by decreasing expiratory flow rates and inducing premature closure of peripheral airways (Sin et al 2004, p.62)." Using a sample of children born at 37 weeks gestation or more from Alberta, Canada, these authors found that children born at a high birthweight (greater than 4500 grams) had an adjusted relative risk of 1.16 for an

emergency room visit for (diagnosed) asthma compared children born at both low birthweights (less than 2500 grams) and normal birthweights. Interestingly, 4.8% of this sample was aboriginal. Thomson (1990) found that Native Indians had a relative risk of 1.47 compared to non-Natives for heavy birthweight (non-Natives were any race-ethnicity reported on the birth registry that were not Native). Indeed, Sin et al (2004) report more aboriginal children in their sample were born at a high birthweight than to normal and low birthweights. The same birthweight pattern is true for AI/ANs (Frank et al 2000). However, this pattern should not introduce a bias towards a positive relationship between high birthweight and asthma because Sin et al (2004) controlled for birthweight in their regression analysis. This research will also control for birthweight.

3.1.2 SOCIOECONOMIC STATUS (SES)

The relationship between SES and asthma has yet to be determined in the literature. The SES variables included in this inquiry are family income, parent education, parent household composition, and status of health care insurance. Some believe low SES poses a risk for asthma and high SES protects against it (Nicholas et al 2005, Dales et al 2002, Erickson et al 2002, Ng Man Kwong et al 2002, Findley et al 2003, Litonjua et al 1999, Schwartz et al 1990). Others believe there is no relationship between SES and asthma at all (Hancox et al 2004, Klinnert et at 2001, Goodman 1999). For example Goodman (1999) used the Add Health data and found no differences in reporting of asthma across five classes of SES.

Poverty

There is a general consensus that poverty has a negative effect on child health. Child asthma is no exception. A number of recent studies confirm that poverty predicts child asthma and respiratory illness. For example, children living in poor neighborhoods in New York City bear the highest disease burden and are four times more likely to be

hospitalized for asthma than children who reside in wealthy neighborhoods (Nicholas et al 2005). Using the Student Lung Health Survey in Canada, Dales et al (2002) found the period prevalence for hospital visits because of asthma were greater for children whose family income was less than $20,000 a year compared to children whose family income was $20,000-60,000 and more than $60,000 annually. Boardman, Finch & Hummer (2001) found that children in the National Maternal and Infant health Survey aged three to four who live below the poverty line had an OR of 1.31 for respiratory disease compared to children who live 200% above the poverty line, in their full logistic regression model. Controlling for race, parent education, parental asthma, and place of residence, Litonjua et al (1999) found no effect of family income on child asthma. Additionally, using the NHANES II, Schwartz et al (1990) found that children living in the lowest family income tercile had a relative odds of 1.7 for asthma compared to children living in the highest family income tercile.

Parental Educational Attainment

The role of parents' highest level of education in childhood asthma is not consistently reported. Some researchers reported a negative correlation, while others reported a positive one. It is clear, however, that parents' education effects prevention/management of child chronic illness (Horner, Surratt & Smith 2002). There are several recent studies that found a negative correlation between parental education and child asthma. In a sample of children from East Harlem in New York City, Findley et al (2003) found that children of parents with less than a high school education had an OR of 1.48 for asthma compared to children of parents who graduated from high school. Dales et al (2002) found that children of parents who did not complete high school (or the Canadian equivalent) had a much higher period prevalence for hospital visits for asthma than children of parents who either completed high school or completed college.

Additionally, Litonjua et al (1999) used The Epidemiology of Home Allergens and Asthma data from Boston to examine the relationship between parental education and child asthma. They found that children of parents with less or equal to a high school education had an OR of 1.2 for asthma compared to children of parents with more or equal to a college education. Von Maffei et al (2001) found that newborns of African American mothers in Connecticut and Virginia had an OR of 1.13 for asthma if their mothers had less than a high school education (compared to newborns whose mothers had graduated from high school).

In contrast, Nelson et al (1997) found that lifetime prevalence of physician-diagnosed asthma increased with mother's educational attainment in a sample from suburban Detroit, Michigan (p.23). Children whose mothers had less than a high school education had a 4.5 lifetime prevalence of asthma; children of mothers who graduated from high school – 7.8; and children whose mother's had more than a high school education – 10.8. Similarly, Boardman, Finch & Hummer (2001) found that children of mothers with less than a high school education were at no greater risk for respiratory problems than children whose mothers completed four years of college (in the full model).

Parent Household Composition

There is no current literature on the relationship between one or both parents living in the household and childhood asthma. There is, however, limited information available on the risk and/or protection that parental marital/cohabitation status poses for child asthma. This research assumes that if both parents are in the home, they are either married or cohabiting.[10] In a study of Los Angeles County, Simon et al (2003) found the

[10] Parent marital status was originally included in the logistic regression analysis, but was found to have no effect, and so was dropped from the analysis. It is assumed that the variable "parent household composition" better captures the relationship between parent stability and child asthma than does "parent marital status."

prevalence rate of childhood asthma was 4.2 percentage points higher for children of divorced, widowed, single parents compared to children of married or cohabiting couples. Von Maffei et al (2001) also found that married parents protected newborns from asthma. In their study of children in two northeastern states, Von Maffei et al (2001) found that newborns of African American mothers who were married had an OR of 0.87 compared to newborns whose mothers were not married.

Source of Insurance

Source of insurance is important for understanding race-ethnic differences in asthma because children without insurance are less likely to manage this chronic illness effectively (Bloomberg et al 2003, Stevens, Sharma & Keston 2003). Also, it is important to note that AI/ANs are among those most likely to have public insurance (Dougherty et al 2005) versus private insurance (likely because of IHS). Few studies have examined the effect insurance status has on childhood asthma. For example, Bloomberg et al (2003) used data from a 10-year period in St. Louis and found that African American children with asthma who had Medicaid or no insurance were at higher risk for hospital readmission than African American children with asthma who had commercial insurance (risk ratio 1.28). Also, using data on children with acute asthma from the Multicenter Airway Research Collaboration, Ferris et al (2001) found that uninsured children had consistently poorer quality of care than insured children with acute asthma. One study examined the relationship between source of insurance and diagnosis of asthma in children. Simon et al (2003) found that children in Los Angeles County who had private insurance had a higher prevalence rate of having been diagnosed with asthma by a health professional than those who had public insurance or where uninsured (7.2; 6.3; 3.3 prevalence rates respectively), suggesting having insurance presents a selectivity issue in terms of diagnosis. This is likely evidence of a selectivity

issue, where children who have the means to access health care (i.e., private insurance) are have a higher chance of being diagnosed with a health condition.

3.1.3 ENVIRONMENTAL FACTORS

A child's environment has a major impact on his/her health. In terms of asthma, the discussion of environment is two-fold: the *physical* environment, and the *family* environment. The data selected for this research does not provide detailed information about the child's physical environment, such as characteristics about the home/house. However, the child's exposure to second-hand smoke can be approximated by looking at parental smoking. The child's family environment can be approximated more closely using parental health behaviors, such as parent use of alcohol, parent self-rated health, and parental obesity. At this time, there are no studies that explore the relationship between parent use of alcohol or parent self-rated health and child asthma.

Parent use of alcohol may prove important, however, because parents who drink heavily have lowered inhibitions and decreased frontal lobe functioning (Child Trends Data Bank 2004). Both of these side effects of intoxication can lead to an unsafe home for children, and a home with risks for asthma (E.g., unclean home [mold, pests], second hand smoke). It is unclear, however, how healthy or light drinking habits affect child asthma. Parent self-rated health provides a glimpse into the child's health environment. While not inclusive of asthma, Waters et al (2000) found that parents self-reporting poor health had an increased odds (OR = 7.5) of reporting their children had poor health.

Parental Smoking

One of the greatest, avoidable risks to child health is second hand smoke, also known as environmental tobacco smoke [ETS] (Feinson & Chidekel 2006, Hawamdeh, Kasasbeh & Ahmad 2003, Dezateux et al 2001, Weitzman, Gortmaker & Sobol 1990). Specifically, ETS is the mixture of sidestream smoke and exhaled mainstream smoke that

pollutes air in the location where tobacco is being smoked (Tutka, Wielosz & Zatońiski 2002, p.325). Approximately 43% of children two months to 11 years live with at least one smoker (Child Trends Data Bank 2006). A study in Jordan estimated that the nicotine dose received by children whose parents smoke is equivalent to their actively smoking between 60 and 150 cigarettes annually (Hawamdeh, Kasasbeh & Ahmad 2003, p.441).

The relationship between ETS and child asthma is well documented, though not consistent. In Los Angeles County, children exposed to environmental tobacco smoke have a prevalence rate of asthma nearly twice that of children who are not exposed (Simon et al 2003). African American newborns that lived with a smoker had an OR of 1.21 for asthma compared to newborns that did not (von Maffei et al 2001). Klinnert et al (2001) found that children aged 6 to 8 in a sample from Denver, Colorado had an unadjusted OR of 1.54 if their mother smoked at least one pack a day. In contrast, Arif et al (2004) found that living with a smoker slightly decreased the odds of having asthma or wheezing in children. Unfortunately, the data selected for this research do not provide information specific to ETS. This information can be approximated, however, by measuring whether or not the child's parent smokes.

Parental Obesity

Obesity is a risk factor for asthma in both adults and children (Arif et al 2004, Bibi et al 2004, Chinn 2003, Schachter et al 2001). For example, Rodriguez et al (2002) found that children in the NHANES II study who had a body mass index (BMI) in the 85th percentile or greater had an OR of 1.94 compared to children with a BMI below the 85th percentile. Also, Flaherman & Rutherford (2006) conducted a meta-analysis of nearly 40 years of research and concluded that children with high body weight, either at

birth or later in childhood, are at increased risk for future asthma, with summary estimates of the effect of high weight ranging from 1.23 to 1.35.

Unfortunately, child BMI is not available in the public use files of the selected data. Parent BMI is available. While parent BMI is not a direct predictor of childhood asthma, it can provide evidence of the kind of nutritional environment in which the child lives (Variyam 2001, Hood et al 2000, Nguyen et al 1996). More importantly, researchers have found that parent obesity is a strong predictor of a child's obesity (Fowler-Brown & Kahwati 2004, Frisancho 2000, Strauss & Knight 1999, Maffeis, Talamini & Tato 1998, Whitaker et al 1997). For example, Strauss & Knight (1999) found that mother's weight status had a significant effect on the risk of the child developing obesity (adjusted risk of 0.55 if the mother had a low BMI [compared to normal weight], 1.48 if she was overweight and 3.69 if the mother was obese).

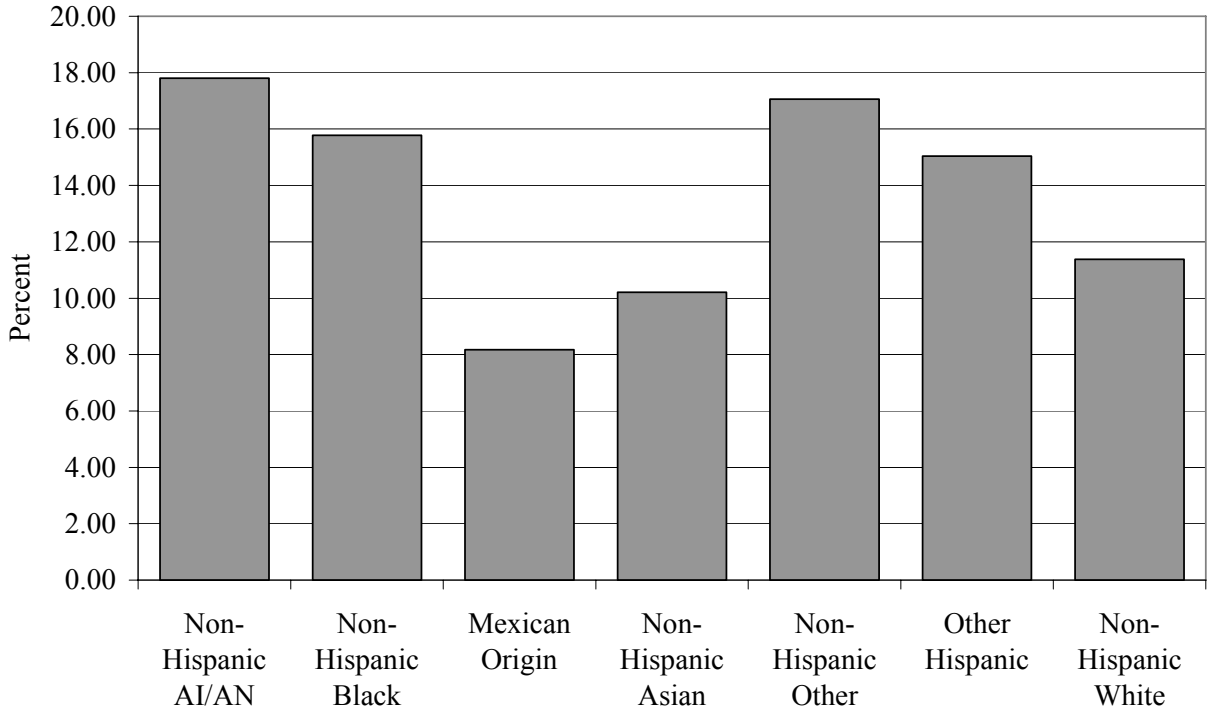
3.1.4 PARENTAL ASTHMA

In an attempt to capture a genetic predisposition to asthma, this research looks at the relationship between parent history of asthma and child asthma, a relationship that has been established as significant in current literature (Raby et al 2005, Rodriguez et al 2002, Klinnert et al 2001, von Maffei et al 2001). Children in the NHANES II data had an OR of 4.00 for asthma if their parents had a history of asthma or hay fever compared to children whose parents did not have such a history (Rodriguez et al 2002). Von Maffei et al (2001) found that newborn of African American mothers who had a history of asthma had an OR of 2.46 compared to newborns whose mothers did not have a history of asthma. In addition, a sample of children aged 6 to 8 in Denver demonstrated a significant association between their asthma and parental asthma (Klinnert et al 2001).

3.2 RESULTS

Overall, AI/AN children in the NHIS data were more disadvantaged than other race-ethnic groups for many of the variables included in this analysis (See Appendix A). In particular, a greater proportion of AI/ANs than children in the other race-ethnic groups identified were born at a low birthweight, are near poor, have parents who are heavy or heavy plus smokers, have parents who are obese or morbidly obese, and have parents with two and three negative health behaviors. They were second to non-Hispanic Blacks in terms of being poor and having parents with fair or poor health, and second to non-Hispanic Others for having parents with a history of asthma. They were also the group that has the highest rate of having been diagnosed with asthma by a health professional (Figure 3.1).

Figure 3.1 Distribution of Asthma by Race-Ethnicity, NHIS 1997-2003 Weighted.



3.2.1 RACE-INCLUSIVE LOGISTIC REGRESSION MODEL

The complete results of this logistic regression are shown in Appendix B. The size of the models prevents their display (in total) in this section. Recall, these models were estimated to evaluate the risk and protection for child asthma across race-ethnicity. In the baseline model, AI/AN children had the greatest odds ratio (OR) of having been diagnosed by a health professional with asthma as compared to non-Hispanic Whites. This finding is illustrated in Figure 3.2. Mexican origin children had the lowest odds for having been diagnosed with asthma (Morales et al 2002). In all of the models, the AI/AN OR for child asthma remained higher than any identified race-ethnic category. Beginning in model two, however, AI/AN risk of child asthma fell below that of non-Hispanic Others. This group was not specified beyond "other race" or "multiple race" in the NHIS dataset documentation for any of the data years.

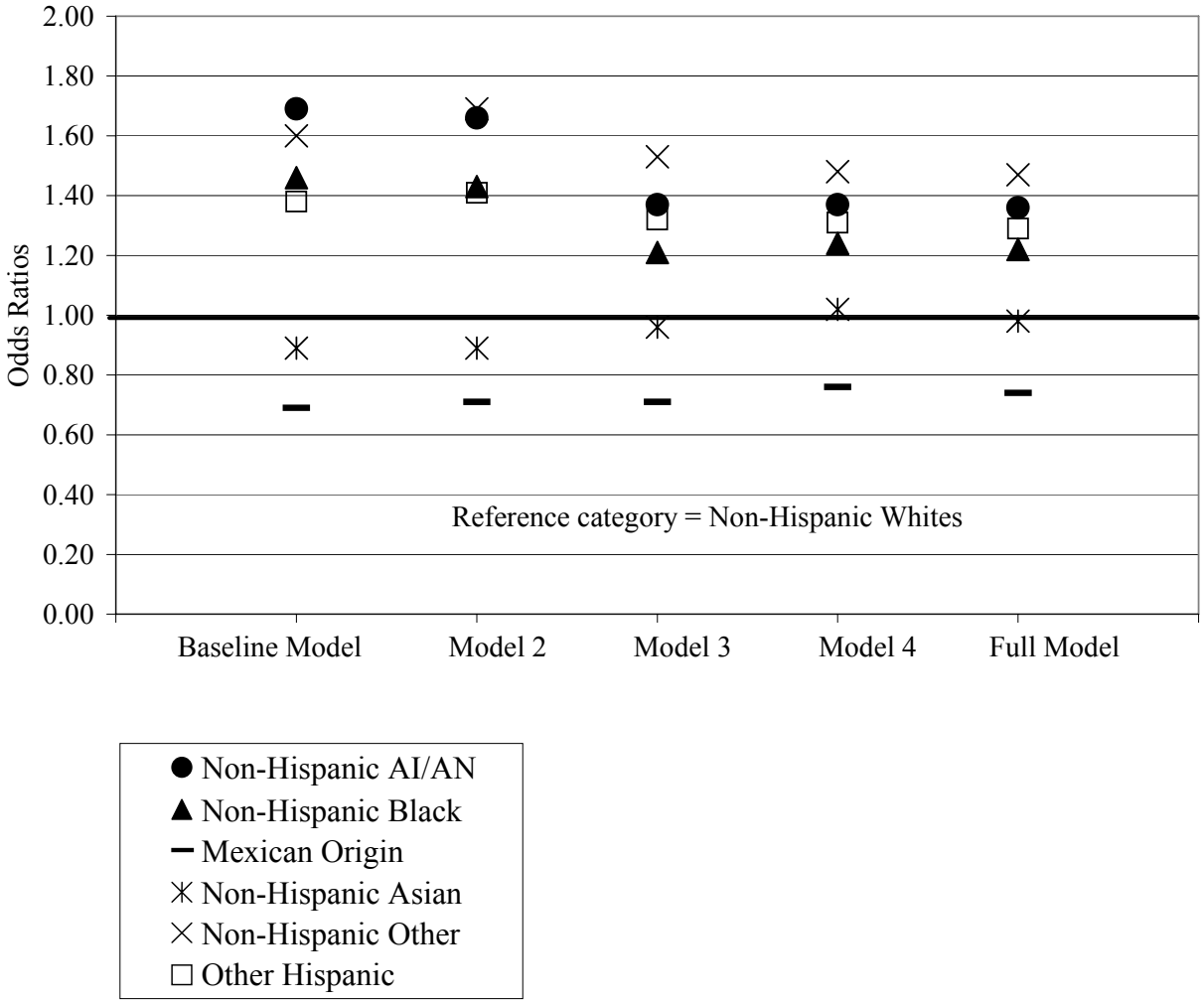
Girls were less likely than boys to have been diagnosed with asthma. Younger children (0-11 years) were less likely than older children (12-17 years) to have asthma. Young age lost some of its protective effect when SES factors were added (compare ORs for age in Model 2 to Model 3). Children born at a *very* low birth weight were at greater risk for asthma than other birthweight categories. These children had an OR of being diagnosed with asthma of more than twice that of children born at a normal weight. Children born at low births weights were also at greater risk than those born at normal weight. The risk of asthma for very low birth weight dropped by 0.15 when SES was added in Model 3, suggesting that environment (economic and health) explains part of the risk for asthma for very low birthweight children.

Children from poor families had a slightly higher OR of being diagnosed with asthma as children from families that were not poor. These ORs, however, were not statistically significant. Unexpectedly, being a child of a near poor family appeared to

have a slightly protective affect. Parent education also yielded unexpected results. It appears that all levels of parental education are protective compared to "some college." Having one parent in the household, however, consistently proved to be a risk for asthma, compared to having both parents in the household. Having government insurance was also a risk for being diagnosed with asthma, versus private insurance. Having no insurance, however, demonstrated a protective effect in this data.

In terms of parental health behaviors, parental drinking did not have an effect on the child being diagnosed with asthma. In contrast, smoking presented a significant risk for child asthma. Indeed, this risk increased as the habitual-level of parental smoking increased. Even being a former smoker presented a risk the child's health. Parental weight that was <u>not</u> normal presented a risk for child asthma, although the results for "underweight" were not significant. In Model 3, a child with a morbidly obese parent had an OR of 1.31 for asthma compared to a child with a parent of normal weight. This variable presented the third highest risk for child asthma (behind birthweight and race-ethnicity). However, once parent history of asthma was added (Model 4), the difference between the risks presented by having an obese or a morbidly obese parent converge. Parent self-rated health that was "excellent" or "very good" protected against child asthma, while "fair" or "poor" parental health presented a risk. In the alternate full model, accumulation of negative health behaviors proved to be a risk, as well. The risk increased as negative behaviors increased. In all of the models, parental history of asthma presented the greatest risk for child asthma.

Figure 3.2. Odds Ratios for Child Having Asthma by Race-Ethnicity,
NHIS 1997-2003 Weighted.



3.2.2 RACE-SPECIFIC LOGISTIC REGRESSION MODELS

Race-specific regression models were estimated to evaluate the risks and protections presented for child asthma across race-ethnicity. Recall most of the variables were condensed to accommodate smaller sample sizes, including age, birthweight (recoded), poverty status, parental education, parental smoking, parental weight status, and accumulation of negative parental health behaviors. Parental use of alcohol was dropped since it was not significant in the race-inclusive model. These models were limited to four identifiable race-ethnic groups: AI/AN, non-Hispanic White, non-Hispanic Black, and Mexican Origin. However, these models yielded few stable estimates for AI/ANs, because of the small sample size. As mentioned earlier, this prevented a meaningful evaluation of how variables that present risk or protection vary across race-ethnicity. In particular, the unstable estimates prevented a meaningful dialogue about the effect of variables for the target population. Thus, only estimates that were stable for AI/ANs, in either the full model or the alternative full model, will be discussed (Table 3.1).

Across race-ethnicity, girls were less likely than boys to have been diagnosed with asthma. It appeared that being a girl is most protective for AI/ANs. Being a child of a parent with at least some college education appears to be protective for AI/ANs, but not for other groups. Having government insurance also provided protection against asthma for AI/ANs, but not for the other groups. This was likely because government insurance was specified as Indian Health Service (IHS) for AI/ANs. Indian Health Service provides a health services to AI/ANs that are members of federally recognized tribes. The results for Mexican origin children for this variable were not significant. Having only one parent in the household was a risk for asthma for all groups, however it was a greater risk for AI/ANs than for all others. Having a parent that is a former or current smoker was a

risk for non-Hispanic Whites and Blacks, but not for AI/ANs. Unexpectedly, this variable demonstrated a protective factor for AI/ANs. As with the race-inclusive model, in these regressions, parental asthma posed the greatest risk for child asthma. It was a much greater risk for AI/ANs that for the other groups.

Table 3.1 Variables with Significance in Race-Specific Models for Child Asthma.

Female

	Full Model		ALT Full Model	
AI/AN	0.62		0.59	++
NH White	0.66	***	0.65	***
NH Black	0.70	***	0.70	***
Mex Orig	0.61	***	0.61	***

At least some college

	Full Model		ALT Full Model	
AI/AN	0.51	++	0.51	++
NH White	1.03		1.02	++
NH Black	1.04		1.04	
Mex Orig	1.32	*	1.33	*

One parent in household

	Full Model		ALT Full Model	
AI/AN	2.23	+++	2.06	++
NH White	1.14	*	1.14	*
NH Black	1.13	++	1.12	++
Mex Orig	1.36	**	1.37	***

Government insurance

	Full Model		ALT Full Model	
AI/AN†	0.46		0.54	++
NH White	1.12	++	1.12	++
NH Black	1.18	++	1.18	++
Mex Orig	1.06		1.06	

Former/current smoker

	Full Model		ALT Full Model
AI/AN	0.55	++	…
NH White	1.17	**	…
NH Black	1.21	*	…
Mex Orig	1.32		…

Parental asthma

	Full Model		ALT Full Model	
AI/AN	4.23	**	4.32	**
NH White	2.89	***	2.91	***
NH Black	2.05	***	2.08	***
Mex Orig	3.08	***	3.11	***

† Reflects IHS as government insurance.
…Variable not included in alternative full model.

3.3 DISCUSSION

A discussion of the results of this study will be couched in a review of the hypothesis from Chapter 1. Overall, the hypotheses projected were supported. As expected, minority children did have higher rates of asthma (see Figure 3.1), save Mexican origin and Asian children. As expected, these latter two groups had the same or lower frequencies of asthma, compared to non-Hispanic Whites. The epidemiologic paradox was evident in the race-inclusive model, where Mexican origin children had the lowest ORs for asthma, suggesting Mexican origin status is protective against asthma. Interestingly, however, this protection did not emerge consistently in the race-specific model. Mexican origin children presented a greater risk for asthma (compared to non-Hispanic Whites and Blacks) if they had one parent in the household and if their parent had a history of asthma.

Also, as expected, boys had a greater frequency of (59.82%) and risk for asthma than girls (asthma rate 40.18%). Becklake & Kauffmann (1999) argue these differences likely attributable to variable lung development in boys and girls. While girls have smaller lungs than boys, they exhibit higher forced expiratory flow rates (p.1121). In addition, older children have higher rates of asthma than younger children (0-3, 11.28%; 4-7, 22.41%; 8-11, 26.08%; 12-17, 40.22%). While it is believed this finding was a result temporal risk exposure, there are no publications that argue length of exposure to risks is correlated with asthma.

Consistent with the "fetal origin hypothesis," this research demonstrated that very low and low birthweight present a risk for asthma (Edwards et al 2003). Normal birthweight, as the referent, is protective of asthma, compared to low birthweight. This was evident in both the race-inclusive and race-specific models (though birthweight is not significant for AI/ANs). Some researchers argue the fetal origins hypothesis is flawed

because the effect of birthweight on health outcomes is mitigated or negated by postnatal centile crossing (Lucas, Fewtrell & Cole 1999). However, this phenomenon cannot be measured using NHIS data.

Only two SES variables proved significant for all children as a risk for asthma, having one parent in the household and having government insurance. These variables were also significant in the race-specific models. The other SES variables, poverty status, parental education, and source of insurance did not produce expected ORs. Instead, the disadvantaged categories of these variables seemed protective against asthma in the race-inclusive model. It may be that children in these categories were not coded as having asthma because they have not been to a health care professional, leaving cases of asthma underreported (across race).[11] Table 3.2 shows that children in families with low SES measures had higher rates of *not* having been to a health professional in over a year. In other words, these variables may have registered as a protection against asthma because they were associated with the child *not* being diagnosed, making this an issue of negative selection rather than positive protection.

The two SES variables that were significant as risk factors for asthma are extremely relevant for AI/ANs. The percentage of American Indian children living with a single parent is higher than among the general population (Sandefur and Liebler 1997). This makes AI/AN children more vulnerable to the risk posed by living with one parent. On the other hand, the risk present by having government insurance is negated by AI/AN children having access to IHS. It is also worth noting that Mexican origin children are at risk for asthma compared to non-Hispanic Whites. This is unusual considering the expected impact of the epidemiological paradox. Some researchers argue that the longer

[11]As noted earlier, asthma is not underreported by race-ethnicity. In a 1994 evaluation study, however, Edwards et al found that asthma was underreported in the NHIS data by approximately 20 to 25% in sample adults (p.22), suggesting it is underreported for all NHIS respondents.

Mexican origin persons remain in the U.S., the more deleterious their health because of negative acculturation. This theory suggests that Mexican origin persons adapt the negative health behaviors of their U.S.–born counterpart, and thus loose the benefits of their previous and positive health behaviors (Cho et al 2004).

Table 3.2 SES by Utilization of Health Services, NHIS 1997-2003 Weighted.

	% Never	% In the Last Year	% More than a Year Ago
Family Income to Poverty Ratio			
Poor (< 1.00)	1.70	86.69	11.62
Near Poor (1.00 - 1.99)	1.71	86.17	12.12
Not Poor (2.00 +)	0.84	91.89	7.27
Parental Educational Attainment			
No High School Diploma	2.83	82.91	14.26
High School Diploma or GED	1.08	88.80	10.11
Some College	0.83	91.23	7.94
College Degree	0.61	93.64	5.75
Source of Insurance‡			
No Insurance	3.82	76.10	20.09
Government Insurance	1.30	89.14	9.55
Private Insurance	0.89	91.26	7.85

These rates reflect responses to "About how long has it been since anyone in the family last saw or talked to a doctor or other health care professional about [child's] health? Include doctors seen while {he/she} was a patient in a hospital."

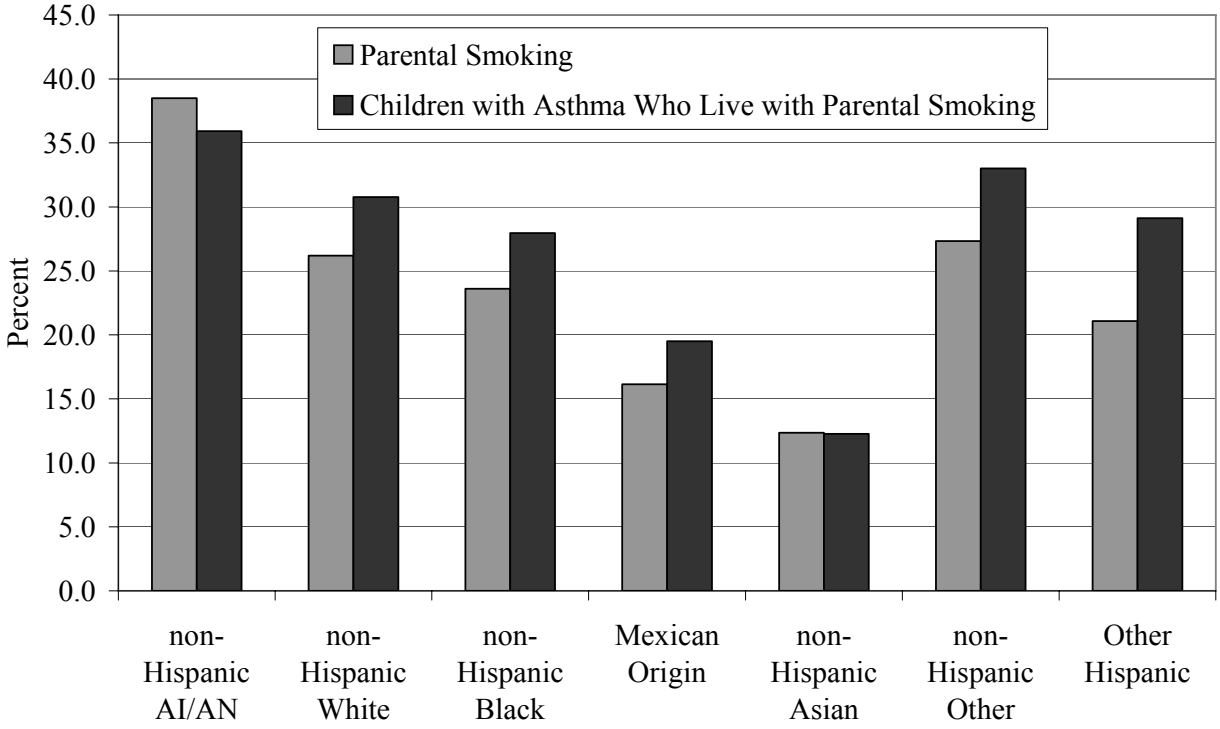
‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

Note: Distributions are shown by row categories such that each row adds to 100%.

Two of the three parental health behaviors presented a risk for child asthma in the race-inclusive model, parental smoking and parental weight status. Recall, the NHIS variable for parental smoking was used as a proxy for child exposure to ETS. It was not possible to tell with these data if the parent actually smokes near the child. Still, the

carcinogenic risk posed by ETS is documented (U.S. Environmental Protection Agency [EPA] Nd). The EPA also identifies ETS as a *causal* agent for child asthma. The National Survey on Environmental Management of Asthma and Children's Exposure to Environmental Tobacco Smoke found that 11% of children aged 6 and under were exposed to ETS on a regular basis in their homes in 2003 (EPA Nd). The EPA further reports that 90% of this exposure is a result of parental smoking. Nearly 39% of AI/AN children in the NHIS data used in this research lived with a parent who smoked (see Figure 3.4). In addition, more AI/AN children with asthma live with a parental smoker than any other race-ethnic group. Even with this evidence, the sample size of AI/ANs was not large enough to yield stable estimates for parental smoking in the AI/AN specific model.

Figure 3.3 Distribution of Parental Smoking and Asthma by Race-Ethnicity, NHIS 1997-2003 Weighted.



Parental weight status was also significant in the race-inclusive model as a risk for child asthma. All of the weight categories presented a risk for asthma compared to normal parental weight. Having a parent that was morbidly obese presented the greatest risk in Model 3, however, once parental asthma was considered, morbid obesity had the same effect as obesity. This is an important finding for the target group as more AI/AN children live with obese parents compared to any other race-ethnic group (see Appendix A). Parental weight was included in this research because child weight was not available. Thus parental weight serves as a proxy for the child's nutritional habits (Nguyen et al 1996, Hood et al 2000). Studies have shown that children learn their dietary and exercise habits from their parents' habits, and this is partly what contributes to child obesity when the parent is also obese (Nguyen et al 1996, Hood et al 2000). This is important for

AI/AN children in that the USDA reported that AI/ANs were not meeting the daily dietary recommendation in terms of grains, fruits, milk, and meat (Basiotis, Lino and Anand 1999).[12] AI/ANs had the greatest OR for parental obesity across the race-specific models, but this finding was not significant.

The accumulation of health behaviors proved to be a risk factor for child asthma in the race-inclusive model. This variable was included to learn if having a parent with more than one negative health behavior would increase the odds of child asthma, compared to having a parent with only one. It appears that it does. The category of two negative health behaviors was most significant, though the OR for this category is slightly lower than the OR for three negative health behaviors. These findings demonstrate that having a parent with no negative health behaviors is protective against child asthma, while having a parent with one to three poses a risk.

Parental asthma was the strongest predictor of child asthma in all regression models. In the race-inclusive model, children with parents who were diagnosed with asthma have an OR of 2.87 in the full model compared to children whose parents do not have asthma. This variable was included in the research to capture a child's genetic predisposition to asthma. The results could be interpreted as a reflection of an inherited trait rather than as due to environmental factors. That is, the ORs for parental smoking did not change much in the full model compared to Model 3. If parental asthma was associated smoking and, thus, child asthma associated with ETS, the full model should have demonstrated this by having either parental smoking or parental asthma drop out of the model. Both variables remained strong predictors of child asthma in the full model. It is possible, however, that both parental and child asthma are associated with traits not measured here, such as environmental exposure to allergens. In the race-inclusive

[12] Interestingly, the USDA reported the AI/ANs sampled did not significantly differ from the rest of the U.S. population regarding indexed diet deficiencies. The actual comparisons were not available for report.

models, parental asthma remained the strongest predictor of child asthma across race-ethnicity. It was a more powerful predictor of child asthma for AI/AN children than for the other race-ethnic groups examined (see Table 3.1). This is not surprising considering more AI/AN children live who have parents with a history of asthma than do non-Hispanic White or Black, or Mexican origin children. Interestingly, parent asthma posed a greater risk for Mexican origin children that for non-Hispanic Whites. Comparatively, parental asthma posed the lowest risk for Non-Hispanic Blacks.

This finding, however, must be interpreted with caution as not all children in this sample had a biological relationship with the repsonding parent. Parental history of asthma is more likely to hold as a genetic proxy for children whose mother was the responding adult, as more mothers (65.3%) were respondents for children with asthma than fathers (34.7%), and a greater proportion of responding mothers had a biological relationship with the child. The percentage distribution of relationship with child (when reported) is as follows (NHIS 1998 – 2003 weighted):

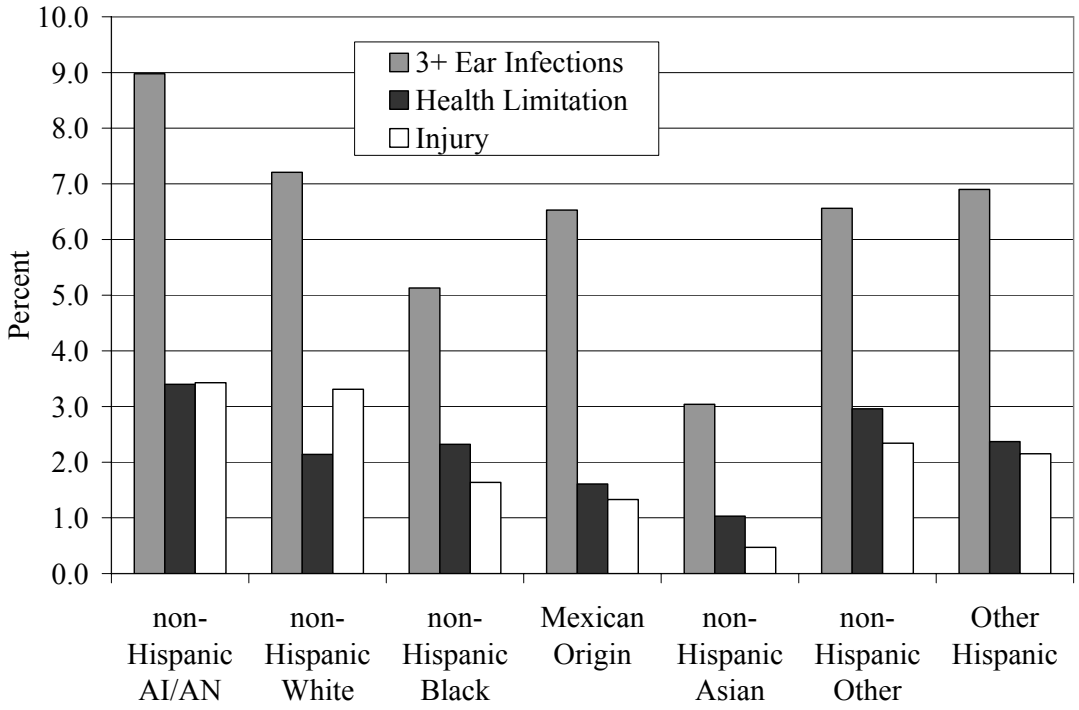
	Biological	Adoptive	Step	Other	Unknown
Mother	95.75	1.85	1.45	0.46	0.48
Father	85.04	2.48	10.60	1.00	0.88

Chapter 4, Ear Infections, Health Limitations, and Child Injury

As mentioned earlier, this chapter will cover the remaining three health outcomes. These health outcomes are relevant for the target population because, like asthma, they are disproportionately experienced by AI/AN children (see Figure 4.1). Condensing the remaining health outcomes is partially due to the logistic regression results not yielding stable estimates for the target population. It is also a pragmatic approach to the literature review. There is very little literature on child health limitations and child injury by sociodemographic variables, such as race-ethnicity, SES, and parental health behaviors. There is more information on ear infections, but not nearly the volume as was the case for child asthma.

This section is divided into subheadings for each outcome. Results from crosstabulations as well as logistic regressions are reported. The regressions are organized the same way the asthma models were, save the exclusion of parental asthma. Overall, more AI/AN children had three or more ear infections, a health limitation, and at least one injury than other race-ethnic groups (Figure 4.1).

Figure 4.1 Distribution of Ear Infections, Health Limitation, and Injury
by Race-Ethncicity, 1997-2003 NHIS Weighted.



4.1 EAR INFECTIONS

Ear infections are known in the medical field as otitis media (OM) (or otitis media with effusion [OME]), an infection or inflammation of the middle ear. Seventy-five percent of children experience at least one ear infection by their third birthday (National Institute on Deafness and Other Communication Disorders [NIDCD] 2006). Almost half of these children will have three or more ear infections during their first 3 years (NIDCD 2006).

4.1.1 LITERATURE REVIEW

Medical literature has long pointed out that AI/AN morbidity due to OM is greater than among the U.S. population in general (Curns et al 2002, CDC 2001). Using IHS data, Curns et al (2002) found that outpatient visits for OM for AI/AN infants were

almost three times that national average. They also found that children 1 to 4 years old had OM visits that were 1.5 times the national average. A number of researchers have found that race-ethnicity is associated with OM (Karevold et al 2006, Vernacchio et al 2004, Lieu & Feinstein 2002, Lanphear et al 1997). However, the research on race-ethnicity and OM is not consistent. Karevold et al (2006) found that Black children (adjusted OR = 1.5) and Asian children (adjusted OR = 1.2) were at greater risk for OM than non-Hispanic White children. In contrast, Vernacchio et al (2004) found these two races were at reduced risk comparatively. Lanphear et al (1997) also found Black children had a lower risk for OM (OR = 0.6), as did Lieu & Feinstein (2002) (non-Hispanic Blacks OR = 0.75). Lieu & Feinstein (2002) also found that Mexican American children had an OR of 0.77 compared to non-Hispanic Whites. Still others found no association between race-ethnicity and OM at all (Woods 2003).

Other child characteristics that have a known association with OM are child age, sex, and birthweight. Several sources advise that younger children are more likely than older children to develop ear infections, including the CDC (2001) (Zeisel et al 2002, Daly et al 1996). Zeisel et al (2002) found a strong and dramatic inverse relationship between age and middle ear infections among African American children in North Carolina. Similarly, Daly et al (1996) found that younger children had an OR of 4.18 for recurrent acute otitis media in children in Minnesota between 1978 and 1984. Numerous studies have investigated the association between sex and otitis media. Table 4.1 demonstrates those findings. For the most part, boys have been demonstrated to have a higher risk for otitis media than girls. The exceptions being Vernacchio et al (2004) who found that girls were at greater risk and Rovers et al (2002) who found that sex had no effect.

Table 4.1 Summary of Studies that Have Evaluated the Association Between Child Sex and Otitis Media.

Authors	Year	Sex	Measure	Result
Karevold et al	2006	Female	Adjusted OR	1.2
Vernacchio et al	2004	Male	Adjusted OR	1.1
Lieu & Feinstein	2002	Male	Unadjusted Risk Ratio	1.03
Zeisel et al	2002	Female	Regression coefficient	–1.60
Rovers et al	2002	…	Correlations	…
Lanphear et al	1997	Male	OR	1.3
Daly et al	1996	Male	OR	1.42

… = no effect

Several studies have also examined the relationship between birthweight and OM, but with inconsistent conclusions. Rovers, de Kok and Childer (2006) conducted a meta-analysis of risk factors for OM using PubMed. They found that low birthweight (less than 2500 grams) was consistently a risk factor in several European countries, the U.S., Canada and Australia. On the other hand, Lieu & Feinstein (2002) found that high birthweight (greater than 4100 grams) was a risk for recurrent ear infections for children in the NHANES III data, with an unadjusted risk ratio of 1.04 compared to normal birthweight. They also found that low birthweight (less than 2500 grams) was protective against recurrent ear infections. Other researchers found that birthweight had no effect on OM (Engel, Streatemans & Zielhuis 2005, Gravel, McCarton & Ruben 1988).

SES has also been associated with OM in children. The variables used to measure SES in recent research are parental education, parental marital status, and insurance status (Karevold et al 2006, Vernacchio et al 2004, Woods 2003, Lieu & Feinstein 2002,

Lanphear et al 1997). Lieu & Feinstein (2002) found that higher parental education presented a risk for ear infections (OR = 1.43 for children of parents with an education above high school, compared to education below seventh grade). Lanphear et al (1997) also found that higher education presents a risk (OR = 1.3 for children of college graduate mothers compared to children of mothers who did not graduate from college). However, Karevold et al (2006) found that children of mothers with less than a college degree in Oslo, Norway were at risk for OM. Similarly, Vernacchio et al (2004) found that Bostonian children of mothers who were not married were at greater risk for OM. Finally, Woods (2003) found that children without insurance were at increased risk for OM.

Environmental tobacco smoke (ETS) also creates a greater risk for OM (Karevold et al 2006, Rovers, de Kok & Schilder 2006, Zeisel et al 2002, Froom et al 2001, Uhari, Mäntysaari & Niemalä 1996). Karevold et al (2006) found that children whose caregiver was a smoker had an adjusted OR of 1.2. In their meta-analysis of OM risk factors, Rovers, de Kok and Schilder (2006) found that parental smoking was a risk factor across several countries. Zeisel et al (2002) also found that children of *non*smoking parents were at a reduced risk for OME (regression coefficient = –1.50 compared to children of smoking parents). In a study of children from North America, the United Kingdom, and the Netherlands, smoking in the home presented a risk for symptoms of acute OM (OR = 1.2). Additionally, another meta-analysis reviewing literature from 1966 to 1994 found that risk of acute OM increased with parental smoking, with a risk ratio of 1.66 (Uhari, Mäntysaari & Niemalä 1996). There was one study, however, that found small exposure to ETS could not produce the ill effects widely claimed in current literature. Denson (2001) argues that poor diet (in response to low SES) is just as likely to produce the poor

health outcomes attributed to ETS (such as abnormal lipid profiles and reduced pulmonary function).

It is also worth noting a reported association between child asthma and OM (Lieu & Feinstein 2002, Corren 2000). As OM and asthma are different physical process, researchers have considered this relationship to be one of comorbidity rather than causality (Thomas et al 2005, Grupp-Phelan, Lozano & Fishman 2001, Lack 2001). For this reason, asthma was not included as a predictive variable for OM in this research. There is also significant evidence that OM is heritable (Casslebrant et al 1999, Ehrlich & Post 1997, Stenstron & Ingvarsson 1997). However, it was not possible to measure parental history of ear infections using the NHIS data. As a result, a genetic proxy was not included in the models predicting ear infections in children.

4.1.2 RESULTS

Recall the NHIS collected data on children with three or more ear infections in the last year. This will be abbreviated as recurrent ear infections (REI) in this section. As previously reported, AI/AN children carry the greatest burden for three or more ear infections across race-ethnic groups in the NHIS data, 1997 to 2003. In the race-inclusive full model, AI/AN was the only race-ethnicity category to present a risk for three or more ear infections compared to non-Hispanic Whites, and this result was not significant. All of the other race-ethnic categories appeared protective against REI compared to non-Hispanic Whites. The remaining child characteristics yielded significant results. Girls were at lower risk for REI than boys. Also younger children were at much greater risk than older children. Indeed younger age presented the greatest risk of all variables included in the model. Children 0 to 3 had an OR of 5.76 compared to children 12 to 17. Even children aged 8 to 11 had a significantly increased risk compared to the referent group. Low and very low birthweight presented a risk for REI,

as well. Very low birth weight presented a greater risk than low birthweight. High birthweight did not appear to have any effect.

Several of the SES variables produced significant results. Children of parents who did not finish high school appeared slightly protected against REI. Children living in homes with only one parent were at greater risk for REI (OR = 1.22) compared to children living with both parents. Insurance status also had an effect. Children with no insurance were protected (OR = 0.86) while children with government insurance were at risk (OR = 1.22). The protective effect of "no insurance" may have been due to small cell sizes. Only 0.37% of the NHIS children were coded as having both REI and no insurance. Interestingly, poverty status had no effect.

All of the parental health behaviors show significant results. Children of parents who did not drink were protected from REI. Children of heavy and heavy plus smoker were at risk. Parent self-rated health produced the most striking results among the family and parent characteristics. Children of parents who reported their health as excellent or very good were protected against REI (OR = 0.78). Children of parents who reported their health as fair or poor had an OR of 1.38. Parental weight status (overweight, obese, and morbidly obese) also presented a risk for child REI. In the alternative full model, having one, two, or three negative parental health behaviors also proved to be a risk for REI. Having three negative parental health behaviors yielded an OR of 1.41.

Most of these patterns held true in the race-specific models. However, several of the variables assumed different effects in the AI/AN model (see Table 4.2). To review all race-specific models for ear infections, see Appendices C-I through C-IV. Very low birthweight and having a parent with fair or poor self-reported health were not risks for REI in the AI/AN model (those these results were not significant). However, their impact was protective. Additionally, not having insurance was not protective in the AI/AN

model, it was a risk. The risk for AI/AN children declined as child age increased. While the OR for child age (0 to 4 and 5 to 11) was above 1.00 and it was much lower compared to other race-ethnic groups.

Table 4.2 Variables with Significant Results in non-Hispanic White Race-Specific Models for 3+ Ear Infections.

Age 0-4

	Full Model		ALT Full Model	
AI/AN	1.04		1.08	
NH White	5.74	***	5.73	***
NH Black	5.21	***	5.20	***
Mex Orig	4.22	***	4.18	***

Age 5-11

	Full Model		ALT Full Model	
AI/AN	1.19		1.18	
NH White	2.42	***	2.41	***
NH Black	2.02	***	2.01	***
Mex Orig	2.25	***	2.24	***

Very low birthweight

	Full Model		ALT Full Model	
AI/AN	0.52		0.53	
NH White	2.24	***	2.23	***
NH Black	1.66	++	1.65	++
Mex Orig	1.19		1.19	

Low birthweight

	Full Model		ALT Full Model	
AI/AN	1.61		1.48	
NH White	1.32	*	1.32	*
NH Black	1.18		1.18	
Mex Orig	1.12		1.12	

One parent in household

	Full Model		ALT Full Model	
AI/AN	1.51		1.53	
NH White	1.18	*	1.18	*
NH Black	1.30	++	1.30	++
Mex Orig	0.98		1.00	

No insurance

	Full Model		ALT Full Model	
AI/AN	1.21		1.28	
NH White	0.83	++	0.83	++
NH Black	0.60	+++	0.59	+++
Mex Orig	0.98		0.97	

Government insurance

	Full Model		ALT Full Model	
AI/AN†	2.46	+++	2.59	+++
NH White	1.30	**	1.30	**
NH Black	1.24	++	1.23	++
Mex Orig	1.07		1.07	

Former/current smoker

	Full Model		ALT Full Model
AI/AN	1.73		…
NH White	1.14	*	…
NH Black	1.12		…
Mex Orig	1.26	+++	…

Table 4.2 Continued.

Overweight

	Full Model	ALT Full Model
AI/AN	0.84 ***	…
NH White	1.15 *	…
NH Black	1.16	…
Mex Orig	0.75 *	…

Obese

	Full Model	ALT Full Model
AI/AN	0.89	…
NH White	1.16 +++	…
NH Black	1.24	…
Mex Orig	1.10	…

Excellent or very good health

	Full Model	ALT Full Model
AI/AN	0.44 ++	0.48 ++
NH White	0.85 *	0.85 *
NH Black	0.62 ***	0.62 **
Mex Orig	0.69 **	0.68 **

Fair or Poor Health

	Full Model	ALT Full Model
AI/AN	0.42	0.44 +
NH White	1.38 **	1.39 **
NH Black	1.21	1.21
Mex Orig	1.38 +++	1.43 +++

One negative outcome

	Full Model	ALT Full Model
AI/AN	…	2.56 +
NH White	…	1.22 **
NH Black	…	1.19 +
Mex Orig	…	0.93

Two to Three negative outcomes

	Full Model	ALT Full Model
AI/AN	…	3.01 +
NH White	…	1.34 ***
NH Black	…	1.42 ++
Mex Orig	…	1.14

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001
† Reflects IHS as government insurance.
… Variable not included in alternative full model.

In the remaining variables, the magnitude of ORs was notably stronger for AI/ANs compared to the other groups reported. The most dramatic differences were for government insurance and accumulation of negative parental health behaviors. The OR for AI/ANs was 2.46 for having government insurance. The nearest OR (1.30) was for non-Hispanic Whites. A similar gap was found between these two groups for one negative parental health behavior. The most notable gap, however, was for children of

parents with two or three negative health behaviors, where AI/AN children had an OR of 3.01 for REI. The next closest OR was for non-Hispanic Blacks, 1.42. Unlike other groups, AI/AN children living in poverty were protected from REI, with an OR of 0.30. This finding did not appear to be due to small cell sizes since over 41% of AI/AN with REI were identified as poor (see Appendix A-I).

4.1.3 DISCUSSION

A number of the hypotheses presented in the first chapter were supported here. To begin with non-Hispanic Black, non-Hispanic Asian, and Mexican origin children all had lower rates of REI than non-Hispanic Whites. These groups also proved to be protective against REI in the race-inclusive model. This finding was consistent with previous research (Vernacchio et al 2004, Lieu & Feinstein 2002, Lanphear et al 1997). Girls were slightly protected against REI compared to boys; however, this effect did not materialize in the race-specific models. As expected, younger age was the strongest predictor of REI for all children and across race-ethnicity. This was not true, however, for AI/ANs. The small cell sizes did not yield stable estimates in this race-specific model. The role of birthweight in predicting REI was not clear in the literature. This research found the same results as Rovers, de Kok and Childer (2006) – that low birthweight and very low birthweight were a risk for REI for all children. Two dimensions of low SES were risk factors for REI, having one parent in the home and having government insurance. The results for having one parent in the home were similar to those of Vernacchio et al (2004) who found that children of unmarried mothers were at risk for otitis media. The other SES variables did not present the risk expected. This may be a selectivity issue. As noted in Table 3.2, a greater propostion of children with low SES deferred doctor visits for more than a year.

As expected, negative behaviors presented a risk for REI. It was not clear how positive parental health behaviors would affect REI. In this research, they were protective against REI. Parental smoking, in particular, was expected to be a risk factor for REI. Parental heavy and heavy plus smoking were a risk. This was consistent with previous literature both in direction and magnitude (Karevold et al 2006, Zeisel et al 2002, Froom et al 2001).

4.2 HEALTH LIMITATION

A health limitation is any condition or disability that limits the child's ability to crawl, walk, run, or play. This is a very general health outcome and may even include some of the other outcomes included in this research (such as asthma). It depends on the perspective of the adult responding to the questions about the child's health. According to Child Trends Data Bank (2005b) 20% of children (aged 5 to 17) in 2004 had a health limitation.

4.2.1 LITERATURE REVIEW

A review of literature on all possible conditions and/or disabilities that may qualify as a health limitation is not possible here. Instead, this section will review literature on health limitations and disabilities (that limit activity) in children, in general, as they pertain to this research. According to Child Trends (2005b) the percentage of children (aged 5 to 17) with one or more limitations was highest in 2004 than in any previously reported year. Several of the variables used in this research have been found to be associated with health limitations. Firstly, birthweight is inversely associated with having a health limitation (the lower the birthweight, the greater the risk for a health limitation) [Elgen et al 2005, McGrath & Sullivan 2002, Avchen, Scott & Mason 2001, O'Callaghan et al 1995]. In a study of births from 1982 to 1984 in Florida, children 12 to 15 years old were found to be at greater risk for a variety of disabilities if they were born

at low and very low birth weights (Avchen, Scott & Mason 2001). For example, children born at very low birthweights had a risk ratio of 4.98 for general disability compared to children born at normal weights.

Health limitations have also been associated with low SES (Newacheck et al 2003, Fujiura & Yamaki 2000, Hogan, Rogers & Msall 2000). Using NHIS data from 1983 to 1996, Fujiura and Yamaki (2000) found that children (aged 3 to 21) who lived in poverty were at greater risk for a health limitation than those who did not (OR = 1.88 in 1996). They also found that living in a single-parent household was a risk (OR = 1.86 in 1996). However, the mechanics of the relationship between poverty and child health limitations is not clear. Porterfield and Tracey (2003) argue that poverty may be the *result* of a child's disability rather than the *cause* of it. Using the NLSY79 Child and Youth Supplement data, they found two important results: (1) children with disabilities are more likely to be born into families in poverty, and (2) the birth of a disabled child significantly increases the risk of a family entering poverty. Poverty is also thought to explain the relationship between health limitations and race-ethnicity. Newacheck et al (2003) found that Black children had a significant risk for a limitation compared to White children when controlling for age, sex, family size, region, and MSA location. However, once the authors added poverty status to the model, the risk for Black children was lower than that for Whites (p.247).

4.2.2 RESULTS

As mentioned earlier, a health limitation is a global measure in the NHIS data and, thus, the outcomes reported here may also be reported elsewhere in this research, such as asthma and injury. Indeed, 44.31% of the children who reported having a health limitation also reported having asthma and 4.70% also reported having an injury (weighted sample). It is impossible to know if the responding adult was referring to these

conditions (asthma or injury) when he or she identified the child as having a health limitation. Ear infections are not likely to limit a child's activity. Still 12.41% of children with a health limitation were also reported to have three or more ear infections in the last year. Overall, however, only 2.12% of children in the NHIS 1997-2003 sample were identified as having a health limitation. This low percentage exacerbates the challenge of researching predictors of health limitation for a population that is small to begin with, like AI/ANs.

Non-Hispanic Black and AI/AN children were at higher risk than non-Hispanic Whites in the baseline model (see Appendix D). However, once SES was controlled for, all but one group (non-Hispanic Other) carry a risk for child health limitation that is lower than non-Hispanic Whites. Very low and low birthweights were a risk for a health limitation. Very low birthweight presented an OR of 4.45 compared to normal birthweight in the full model. Interestingly, high birthweight appears to be protective against a health limitation. Other characteristics of the child that were significant were sex and age. Girls were at lower risk for a health limitation than boys (OR = 0.90). The risk for a health limitation increased incrementally with age and all categories of younger children were at lower risk than children 12-17. Being poor or near poor also presented a risk for health limitation compared to not being poor (ORs = 1.35 and 1.27, in the full model, respectively). Other SES variables that produced substantive results were parent household composition and source of insurance. Children who live with only one parent had an OR of 1.13 for a health limitation compared to those who live with both parents. Also, children with government insurance had an OR of 1.46 compared to those with private insurance.

Several parental health behaviors were also significant predictors of a child health limitation. Children of parents who were heavy smokers, in fair or poor health, or

overweight or obese were at higher risk for a health limitation. The most significant of these variables were parents having fair or poor self-rated health and parental morbid obesity. The accumulation of parental behaviors also proved to be significant, with children of parents with two negative health behaviors having the greatest risk (compared to the other cumulative categories).

The race-specific models were less enlightening. This is almost certainly because of small cell sizes. Table 4.3 shows the variables that were significant in the non-Hispanic White race-specific model (the largest race group). To review all race-specific models for health limtation, see Appendices D-I through D-IV. Small cell sizes particularly affected the race-specific model for AI/ANs (see Appendix A-I). Note that many of the ORs seem counterintuitive in Table 4.3, such as age, very low birthweight, and cumulative negative parental health behaviors. Non-Hispanic Blacks are at a disadvantage compared to other groups in that several of the variables that are protective for others are not protective for them. These include age 5 to 11, high birthweight, and no high school diploma. Consistent with the epidemiologic paradox, Mexican origin children have roughly the same risks and protections as do non-Hispanic White children. The paradox is particularly evident in the very low birthweight ORs, where Mexican origin children have an OR lower than both non-Hispanic Whites and non-Hispanic Blacks.

Table 4.3 Variables with Significant Results in non-Hispanic White Race-Specific Models for Health Limitation.

Age 0-4

	Full Model		ALT Full Model	
AI/AN	0.24	++	0.20	++
NH White	0.44	***	0.44	***
NH Black	0.55	*	0.55	*
Mex Orig	0.68	+++	0.67	++

Age 5-11

	Full Model		ALT Full Model	
AI/AN	0.24	+++	0.20	+++
NH White	0.58	***	0.57	***
NH Black	1.04		1.03	
Mex Orig	0.83		0.82	

Very low birthweight

	Full Model		ALT Full Model	
AI/AN	0.00	***	0.00	***
NH White	4.72	***	4.64	***
NH Black	3.87	***	3.85	***
Mex Orig	3.16	*	3.14	*

Low birthweight

	Full Model		ALT Full Model	
AI/AN	1.63		1.47	
NH White	1.89	***	1.88	***
NH Black	1.69	+++	1.64	++
Mex Orig	0.95		0.95	

High birthweight

	Full Model		ALT Full Model	
AI/AN	0.47		0.42	
NH White	0.76	*	0.77	*
NH Black	1.31		1.34	
Mex Orig	0.87		0.88	

No high school diploma

	Full Model		ALT Full Model	
AI/AN	0.30	++	0.29	
NH White	0.70	+++	0.70	*
NH Black	1.34		1.33	
Mex Orig	1.00		0.99	

Overweight

	Full Model		ALT Full Model
AI/AN	0.51		…
NH White	1.20	++	…
NH Black	1.05		…
Mex Orig	1.39		…

Obese

	Full Model		ALT Full Model
AI/AN	0.47		…
NH White	1.22	++	…
NH Black	1.50	++	…
Mex Orig	1.39		…

Table 4.3 Continued.

Excellent or very good health

	Full Model		ALT Full Model	
AI/AN	0.32		0.32	
NH White	0.43	***	0.43	***
NH Black	0.84		0.82	**
Mex Orig	0.44	***	0.44	***

Fair or Poor Health

	Full Model		ALT Full Model	
AI/AN	2.82		2.24	
NH White	1.45	*	1.45	*
NH Black	2.20		2.23	
Mex Orig	1.42		1.42	

One negative outcome

	Full Model		ALT Full Model	
AI/AN	…		0.40	
NH White	…		1.24	+++
NH Black	…		1.21	
Mex Orig	…		1.60	++

Two or three negative outcomes

	Full Model		ALT Full Model	
AI/AN	…		0.39	
NH White	…		1.38	*
NH Black	…		1.84	+++
Mex Orig	…		1.29	

… Variable not included in alternative full model.

4.2.3 DISCUSSION

Many of the results for child health limitation were consistent with results reported in current literature, as well as with the hypotheses from Chapter 1. For both the race-inclusive and the race-specific models, most of the ORs are intuitive. This is not the case for the AI/AN race-specific model. As with REI, it is believed small cell sizes did not yeild stable estimates in this model. To begin with, very low and low birthweights presented a risk for health limitations, while normal birthweight appeared to be protective. This demonstrated the noted inverse relationship between birthweight and health limitations found in previous literature (Elgen et al 2005, McGrath & Sullivan 2002, Avchen, Scott & Mason 2001, O'Callaghan et al 1995). Unexpectedly, high birthweight also appeared to be protective. Also, low SES presented a risk for child health limitation. Consistent with Fujiura and Yamaki (2000), this research also

demonstrated that being poor or near poor is a risk for health limitation. This research is consonant with literature in that children living with only one parent are also at greater risk (Fujiura & Yamaki 2000, Hogan, Rogers & Msall 2000).

Admittedly, the protective effect of low parental education does not make substantive sense. Indeed, none of the parental education outcomes appears to make sense. If low education only was protective against a health limitation, it could be argued that such parents have a different perspective on what constitutes a health limitation (recall the limitations are "self-reported" by the responding adult, not diagnosed by a health care professional). However, having a college degree is also protective, so this hypothetical argument is not consistent with the results.

As hypothesized, girls and younger children were protected from health limitation compared to their respective referent categories. There is no literature that speaks directly to sex and age and their associations with health limitations. It may be that the literature referenced in Chapter 1 holds true here for sex. This literature suggested that boys engage in riskier behavior, placing them at greater risk for negative health outcomes than girls. Older children were expected to have a higher risk for a health limitation because they have had longer to be exposed to experiences and agents that might induce or cause such a limitation.

There is no literature available to help explain the relationship between parent self-rated health and parental weight status and child health limitation. It may be that parents with health habits provide a healthy environment for their children and vice versa. Perhaps that is why these behaviors have the reported effects. There is at least one article which documented that parent smoking (ETS) was associated with child health limitation. Hogan, Rogers & Msall (2000) found that children with limitations were likely to live in

homes with a smoker and, further, these children are also more likely to spend *more* time at home and thus have increased exposure to the ETS (p.1046).

4.3 INJURY

Child injuries are a national concern because they are preventable (CDC Injury Center 2006). The data included in this research were limited to those injuries that required medical attention (even if that attention was a phone call to a health care provider).

4.3.1 LITERATURE REVIEW

Child injury was included in this research because it is an important health issue for the target population. More AI/AN children die from accidental injury than children from other race-ethnic groups (National Safe Kids Campaign [NSKC] 2003). Injuries account for 75% of all deaths among AI/AN children (CDC 2003). There is little information on child injury and what child, parent, or environmental characteristics may explain differences in injury across race-ethnicity. More children under one year are fatally injured than other age groups (Wallace, Cody & Mickalide 2003). The risk of death from injury decreases as children age (NKSC 2003). This trend is attributed to young children's poor impulse control, poor judgment, natural curiosity, and lack of fear (NKSC 2004). More boys die from injury than girls each year (NKSC 2003, 2004). In 2000, the fatal unintentional injury rate for boys was 1.5 times that for girls (NKSC 2003).

There is strong evidence that SES is associated with child injury (Simpson et al 2005, NSKC 2004, Faelker, Pickett & Brison 2000, Anderson et al 1998, Durkin et al 1994). A recent study of junior high and high school students found that lower SES was associated with increased risk for hospitalization due to fighting, while higher SES was associated with increased risks for sports/recreational injuries (Simpson et al 2005)

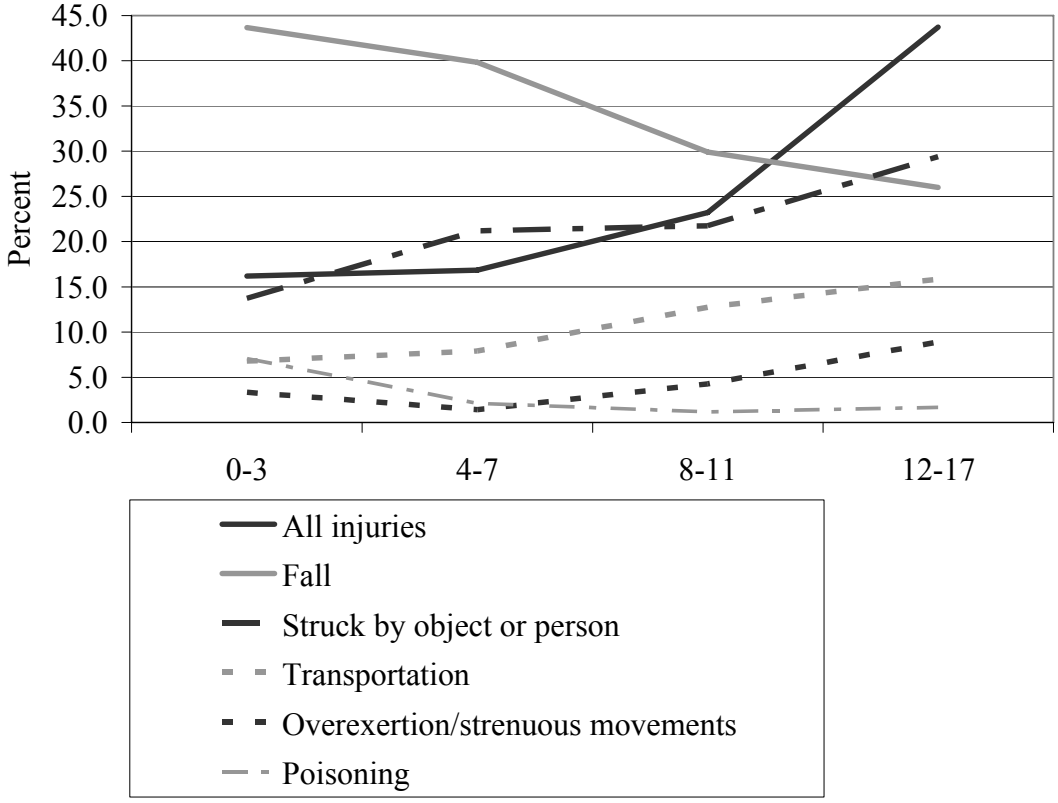
Another study of Canadian children found that SES gradients identified in adult morbidity and mortality held true for child injury. Specifically, the authors reported that gradients of SES were associated with injuries in the home, injuries from recreation (play), and falls (Faelker, Pickett & Brison 2000).

There is also a documented association between race-ethnicity and child injury, though this is believed to be more a function of SES than ethnicity (Simon et al 2006, NSCK 2004, Anderson et al 1998). As mentioned earlier, AI/AN children carry a disproportionate burden for child injury than other groups. Black children have the second highest rate of fatalities due to injury (Wallace, Cody & Mickalide 2003). A recent study also found that African American children (age 18 and under) were hospitalized for injury more so than non-Hispanic White and Hispanic children (18.3 per 100 person years vs. 16.2 and 9.9 respectively). Other evidence available on race-ethnicity suggests Hispanicity protects against child injury (Simon et al 2006, Anderson et al 1998). Anderson et al (1998) found that poor Hispanic children were at lower risk for injury compared to poor non-Hispanic Whites in Orange County, California.

4.3.2 RESULTS

The distribution of injury by sex and age were as expected (weighted sample). Boys carried a greater burden of injury than girls. The data showed 60.48% of the injuries reported were for boys versus 39.52% for girls. Older children also carried a greater burden of injury (0–3, 16.2%; 4–7, 16.85%; 8–11, 23.22%; 12–17, 43.74%). The distribution of injuries by race-ethnicity essentially reflects the distribution of race-ethnicity for the NHIS child sample: non-Hispanic AI/AN, 0.88%; non-Hispanic White, 80.21%; non-Hispanic Black, 7.86%; and Mexican origin, 7.70%. The distribution of injury by SES demonstrated that children who are “not poor” carry the greater burden (poor, 13.78%; near poor, 20.19%, and not poor, 66.03%).

Figure 4.2 Cause of Injury by Age, NHIS 1997-2003 Weighted.



Note: There are two denominators represented in Figure 4.2. The denominator for line "all injuries" was the total sample. The denominator for remaining lines was children whose responding adult reported at least one injury.

There were no major differences in cause of injury or place of injury by sex. There were a few striking changes by age, and those are illustrated in Figure 4.2 (shown are changes of more than five percent between the youngest age and oldest age). It is not possible to completely compare causes of and places of injury across race-ethnicity. This is because the responses offered by adults speaking for AI/AN children provided limited responses (see Table 4.4 – a full listing of injuries in is Appendix F). In both categories (cause and place), the respondents for AI/AN children answered "other" more than the three comparison groups. The other clear differences appear were: (1) a smaller proportion of AI/AN children were reported to have received their injury in an auto accident; (2) a greater proportion of AI/AN children were reported to have been injured

outside the home, at a sports facility, a farm, or a park; and (3) a smaller proportion of AI/AN children were reported to have received their injury at school or in the street.

NHIS recommended the injury files be used with caution because of underreporting (NCHS 1997–2003b) . It appears this caution is well-deserved. The race-ethnic results from the logistic regression models are counter intuitive (see Appendix E). According the race-inclusive model, AI/AN children have about the same risk for injury as do non-Hispanic White children. Additionally, all other minority groups have comparatively lower risk for injury. Several of the findings are intuitive. Girls are at lower risk than boys for injury and younger children are at lower risk for injury than older children. Higher SES did not prove to be protective against child injury. The only protective SES factor was having both parents in the household (having only one parent resulted in an OR of 1.17).

Table 4.4 Percent Distribution of Cause and Place of Injury by Race-Ethnicity: Limited to responses provided by AI/ANs, NHIS 1997-2003 Weighted.

	non-Hispanic AI/AN	non-Hispanic White	non-Hispanic Black	Mexican Origin
Cause of Injury				
Transportation	7.60	11.54	18.67	14.66
Fall	34.04	31.79	29.60	39.37
Overexertion/strenuous movements	2.72	6.21	2.95	2.35
Struck by object or person	20.10	24.14	23.86	17.28
Cut/pierce	6.89	7.80	5.62	7.35
Other	28.66	11.04	13.01	6.75
Place of Injury				
Outside home	43.21	22.44	21.11	27.17
School	4.67	17.47	16.25	15.81
Street/highway	4.32	8.17	15.88	8.62
Sport facility, ball field, playground	19.67	11.44	4.06	4.93
Farm	7.01	0.40	0.00	1.26
Park/recreation area	9.52	4.29	4.19	6.28
Other	11.59	3.77	6.00	3.91

Unexpectedly, parental health behaviors were significant predictors of child injury. Children whose parents did not drink were less likely to have an injury, as were children of parents who reported excellent or good health. Children of smoking or obese parents were at higher risk for injury. The same was true of children of parents with one to three negative health behaviors.

The race-specific models did not yield many significant results. This is likely due to the small number of injuries available for the child records in the NHIS data. Only 2.74% of the children in this data were reported to have at least one injury in the last three months. The variables shown in Table 4.5 are those that were significant for non-Hispanic Whites. Since this comprises the largest group in the data, the table shows the

maximum number of variables. Small cell sizes for AI/ANs produce some bizarre results, such as for age 0–4 and for parents reporting excellent of very good health (see Appendix A-I).

Table 4.5 Variables with Significant Results in non-Hispanic White Race-Specific Models for Child Injury.

Female

	Full Model		ALT Full Model	
AI/AN	0.94		2.02	
NH White	0.65	***	0.65	***
NH Black	0.69	+++	0.69	+++
Mex Orig	0.62	+++	0.62	+++

Age 0-4

	Full Model		ALT Full Model	
AI/AN	0.00	***	0.00	***
NH White	0.57	***	0.57	***
NH Black	0.62	++	0.63	++
Mex Orig	0.80		0.81	

Age 5-11

	Full Model		ALT Full Model	
AI/AN	0.70		0.73	
NH White	0.68	***	0.68	***
NH Black	0.75	++	0.76	
Mex Orig	0.79		0.80	

At least some college

	Full Model		ALT Full Model	
AI/AN	1.77		1.79	
NH White	1.22	*	1.26	*
NH Black	1.24		1.27	
Mex Orig	1.03		1.03	

One parent in household

	Full Model		ALT Full Model	
AI/AN	1.26		1.18	
NH White	1.23	*	1.20	+++
NH Black	1.35	++	1.37	++
Mex Orig	0.92		0.94	

Overweight

	Full Model		ALT Full Model
AI/AN	1.07		…
NH White	1.17	++	…
NH Black	1.00		…
Mex Orig	0.92		…

Obese

	Full Model		ALT Full Model
AI/AN	1.23		…
NH White	1.19	++	…
NH Black	1.01		…
Mex Orig	0.88		…

Excellent or very good health

	Full Model		ALT Full Model	
AI/AN	8.10	+++	9.52	+++
NH White	0.82	*	0.83	+++
NH Black	0.80		0.83	
Mex Orig	0.97		0.99	

Table 4.5 Continued.

One Negative Parental Health Behavior				Two Negative Parental Health Behaviors			
	Full Model	ALT Full Model			Full Model	ALT Full Model	
AI/AN	…	2.43		AI/AN	…	2.90	
NH White	…	1.16	++	NH White	…	1.38	**
NH Black	…	1.17		NH Black	…	1.59	++
Mex Orig	…	1.24		Mex Orig	…	1.10	

… Variable not included in alternative full model.

There was one other variable that produced a significant OR for AI/ANs not listed above. Children of parents with fair or poor health demonstrated an OR of 24.57 ($p<.01$) in the full model and 25.88 ($p<.01$) in the alternative full model. The referent category was "good" parent self-rated health. It is not clear if these results are reliable estimates since the ORs produced by the other comparative category were not intuitive and not at all consistent with the other race-ethnic categories. To review all race-specific models for child injury, see Appendices E-I through E-IV.

4.3.3 DISCUSSION

Race-ethnicity did not demonstrate the expected association with child injury. While more AI/AN children suffered a higher proportion of injuries than any other group, all other minorities had lower rates of injury than non-Hispanic Whites (Appendix A). The race-inclusive model did not offer new insight to this unusual finding. All minority race-ethnic groups had a lower risk for injury than the referent (non-Hispanic Whites). These findings are likely the result of small cell sizes, rather than a true reflection of the association between race-ethnicity and child injury.

As hypothesized, more injuries were attributable to boys than to girls. This is likely because boys engage in riskier behavior than girls (Morrongiello & Rennie 1998).

Compared to boys, girls have a low OR for injury. The role of age in child injury was left unclear in the hypotheses for this research. While evidence suggests that very young children are more likely to be fatally injured, there is no information on what age group bears more nonfatal injuries (NCKS 2004, 2003, Wallis, Cody & Mickalide 2003). This research demonstrates that older children have a larger proportion of injuries than do younger children. This is probably a function of exposure to risk. That is, older children are probably more active than very young children. For example, team sports are generally *not* advised for children under age 7 because motor skills and judgment are not well developed (Mayo Clinic 2006). The race-inclusive model demonstrated that risk is positively correlated with age.

Having a parent who abstains from alcohol appears to be protective against injury. This seems to make sense in that parents who consume too much alcohol would be less able to supervise their children and more likely to engage in reckless behavior, such as driving under the influence, etc. (Child Trends Data Bank 2004). It is not clear why parental health behaviors present a risk for child injury. It may be that parents with positive health behaviors consciously or unconsciously teach their children this behavior. Perhaps it is the case that parents with habitual (or even addictive) behaviors, like drinking, are less likely to supervise their children closely (Children of Alcoholics Foundation Nd). The SES variables for poverty, parent education, and insurance did not prove to be a significant predictor of child injury in this research. Having one parent in the household was a risk for injury. Perhaps this is another issue of supervision. It is more difficult for a child living with one parent to receive adequate supervision than it is for a child living with two parents (Tsushima & Gecas 2001).

Chapter 5, Conclusion

This research focused on AI/AN children in an attempt to fill a gap in public health literature on the association between child health and selected child characteristics, socioeconomic characteristics, and environmental factors. In terms of descriptive analyses, this objective was accomplished. It was not surprising to find that a greater percentage of AI/AN children were reported as having these selected outcomes than children from other race-ethnic groups. These outcomes were selected because they have been identified as health concerns for the AI/AN population.

This research estimated significant ORs for child asthma for AI/ANs, but not for the other health outcomes. This was likely because of the small number of AI/ANs in the NHIS data. This section will review the specific aims and the theoretical approach to this research. In addition, this section will review limitations of this research as well as findings produced here that were not investigated in previous literature.

5.1 SUMMARY

The specific aims of this project were listed in the first chapter. These aims identified three main goals of this project. These goals have been reported separately by health outcomes, using the results of the race-inclusive models. The first goal was to evaluate the predictive power of child characteristics for the selected health outcomes. All of the child characteristics proved to be significant predictors of health. Figure 5.1 demonstrates the effect of race-ethnicity on the selected health outcomes (*non*significant ORs in grey). Most of the other race-ethnic categories in the race-inclusive model were significant for each outcome. Of the identifiable race-ethnic groups, AI/ANs have the greatest risk for each of the outcomes (though that risk is only significant for asthma). Sex was also a significant predictor of health. In all of the race-inclusive models, girls

were at a significant, lower risk than boys for each of the outcomes, as well (ORs = 0.65 asthma, = 0.95 ear infections, = 0.90 health limitation, and = 0.68 for injury). Age was also a significant predictor of health. For all outcomes, save ear infections, young age was protective from poor health. In the case of ear infections, however, young age at the greatest risk for poor health (as expected). Finally, birthweight also proved to be a significant predictor of child health. Figure 5.2 illustrates how birthweight performed for each outcome (*non*significant ORs in grey vs. black).

Figure 5.1 Odds Ratios from Race-Inclusive Full Models by Health Outcome and Race-Ethnicity, NHIS 1997-2003 Weighted.

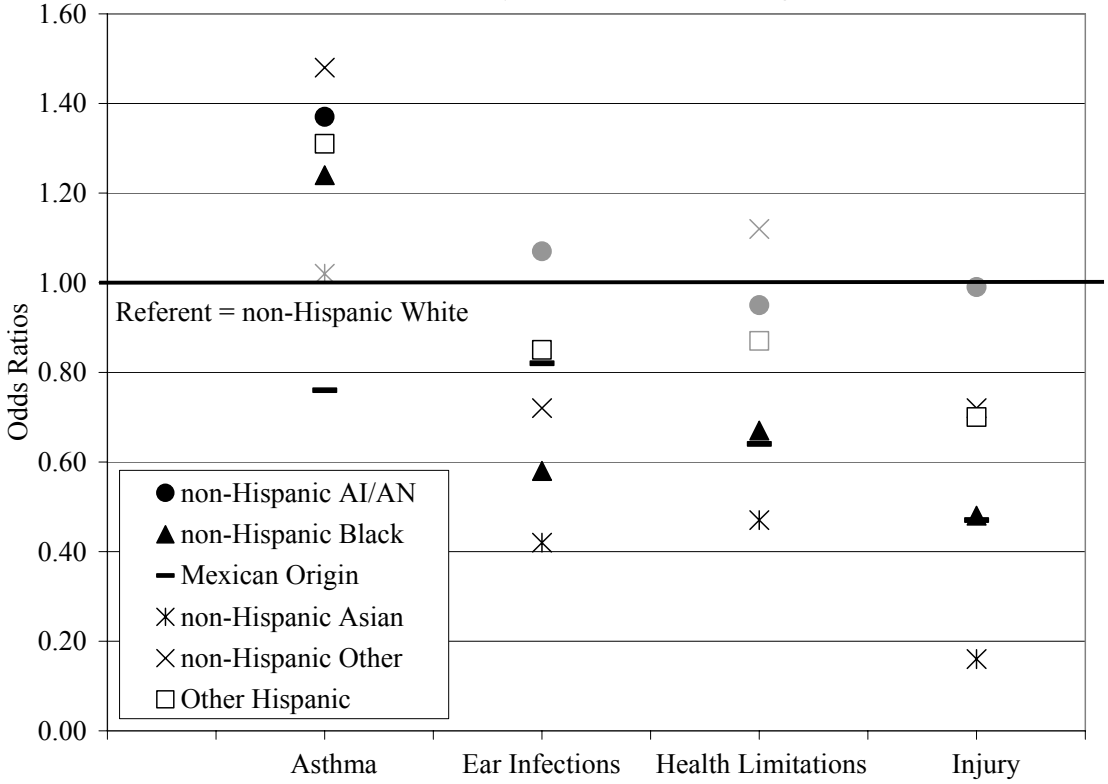


Figure 5.2 Odds Ratios from Race-Inclusive Full Models by Health Outcome and Birthweight, NHIS 1997-2003 Weighted.

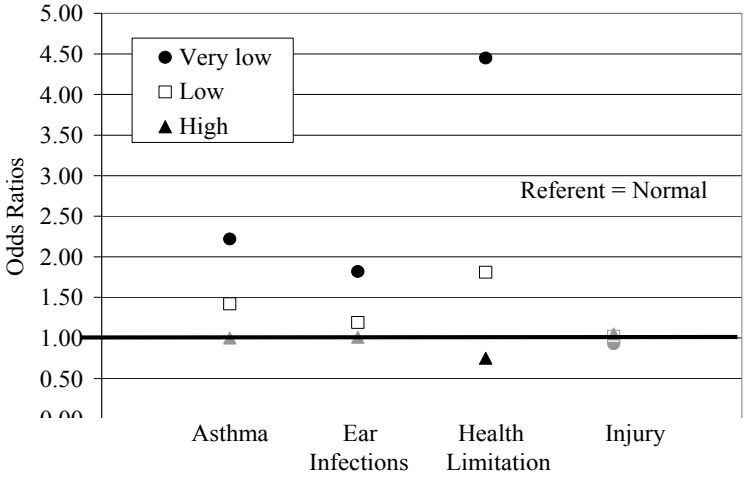


Figure 5.3 Odds Ratios from Race-Inclusive Full Models by Health Outcome and Parental Smoking, NHIS 1997-2003 Weighted.

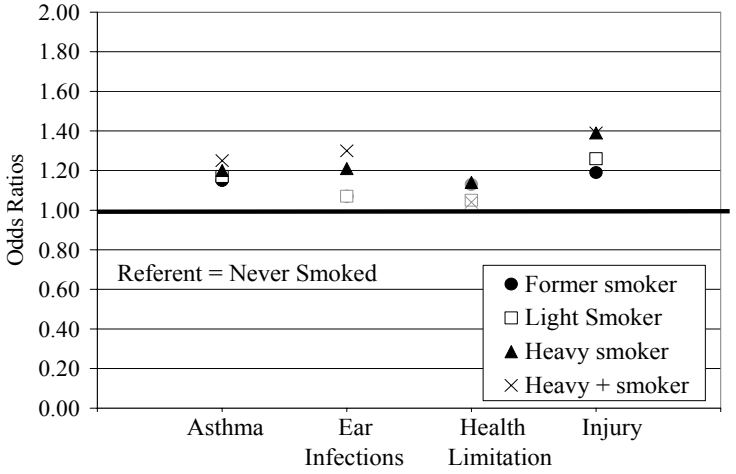


Figure 5.4 Odds Ratios from Race-Inclusive Full Models by Health Outcome and Parental Weight, NHIS 1997-2003 Weighted.

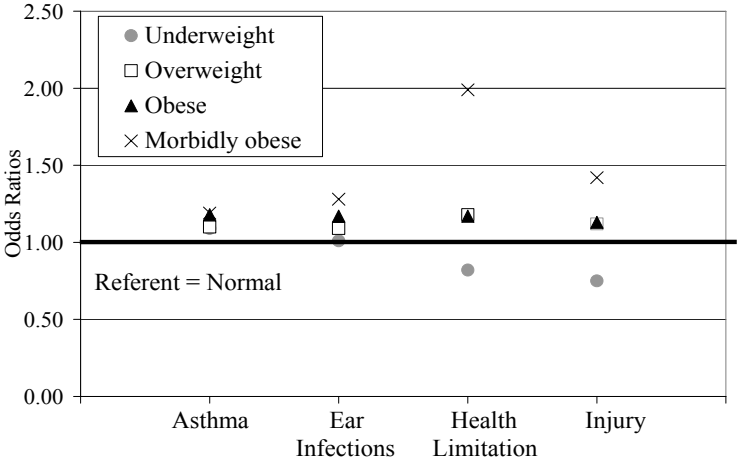
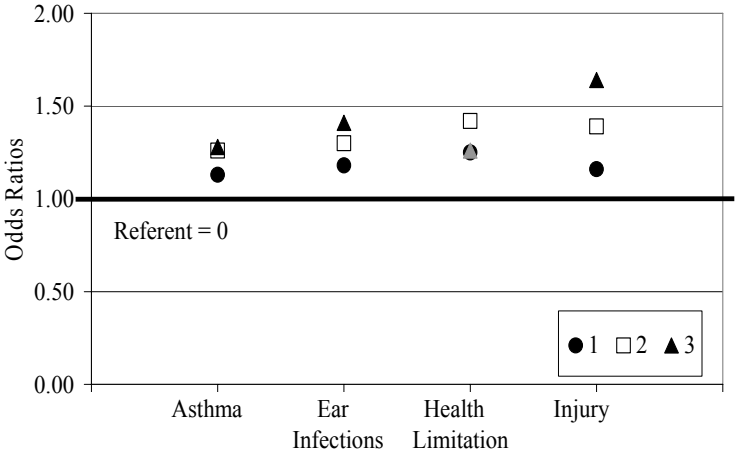


Figure 5.5 Odds Ratios from Race-Inclusive Full Models by Health Outcome and Acumulation of Negative Parental Health Behaviors, NHIS 1997-2003 Weighted.



The second specific aim of this project was to determine what socioeconomic (SES) characteristics predicted child health. Table 5.1 lists the SES variables from the race-inclusive models (*non*significant ORs in grey). Only one variable consistently predicted poor child health, having one parent in the household. As mentioned earlier, this is likely a selectivity issue, where a greater proportion of children of low SES did not visit a health professional in the last year, thus were not given the opportunity to be diagnosed with the selected health outcomes.

Table 5.1. SES Variables that Predict Child Health: ORs from Race-Inclusive Models.

	Asthma	**Ear Infections**	**Health Limitation**	**Injury**
Family Income to Poverty Ratio				
(Referent = Not poor)				
Poor	1.04	0.99	1.35	1.00
Near poor	0.93	0.96	1.27	1.09
Parent Educational Attainment				
(Referent = Some college)				
No high school diploma	0.86	0.91	0.66	0.75
High school diploma or GED	0.93	1.01	0.85	0.80
College degree	0.91	0.99	0.80	1.00
Parent Household Composition				
(Referent = Both parents in home)				
One parent in household	1.21	1.22	1.13	1.17
Source of Insurance				
(Referent = Private insurance)				
No insurance	0.87	0.86	0.85	0.92
Government Insurance	1.11	1.22	1.46	0.91

Appendix G offers a thorough review of how utilization varies by race-ethnicity. In those instances where the distribution of having seen a health professional more than a year ago is negatively associated with higher SES, the issue of selectivity is salient. There is one instance, however, where selectivity did not appear to explain why certain SES variables did *not* present a risk for poor health outcomes in children. The distribution of having seen a health professional more than a year ago showed little variation for AI/ANs. This even distribution was not represented in the OR estimates, however, likely because of the small cell sizes that have consistently presented a challenge to this research. This appendix also provides additional evidence for the epidemiological paradox. Across all categories of SES, a greater proportion of Mexican origin children had *not* seen a health professional in the last year. Still, in the race-inclusive models, this group demonstrated ORs that were lower than non-Hispanic Blacks, as well as non-Hispanic Whites.

The final specific aim was to determine what environmental (parental) characteristics affect the selected child health outcomes. Four of these variables proved to be robust across the varied outcomes, parental smoking, parental weight status, parent self-rated health, and accumulation of negative parental behaviors. The ORs produced by most of these variables are illustrated in Figures 5.3 through 5.5 (*non*significant ORs in grey). It was expected that parental smoking would impact child asthma and ear infections. It was surprising to find that having a parent who smoked also affected having a health limitation and having at least one injury. Additionally, the impact of parent self-rated health was not expected. It was not clear how this variable would be associated with child health as there is very little literature on this. As mentioned earlier, this may be related to children learning health behaviors from their parents. Children of parents who reported excellent or very good health were consistently protected from poor

health. Children of parents who reported fair or poor health were consistently at risk for poor health:

	Asthma	Ear Infections	Health Limitation	Injury
Excellent or very good	0.82	0.78	0.49	0.86
Fair or poor	1.13	1.38	1.55	1.11

It was also a surprise to find that parental weight predicted all of the outcomes. It was expected that parental weight might predict asthma because child obesity is a risk for asthma and parental obesity is suggestive of the child's nutritional environment (Fowler-Brown & Kahwati 2004, Kanda, Kamiyanna & Kawaguchi 2004, Hood et al 2000, Strauss & Knight 1999, Whitaker et al 1997, Nguyen et al 1996). Finally, the predictive power of the accumulation of parental negative behaviors was unknown. No previous literature used this kind of marker for parental health. Though no significant across all health outcomes, having 1 to 3 negative health behaviors consistently presented as a risk for poor child health. For asthma, ear infections, and injury, the risk of poor health increased incrementally with the number of negative health behaviors. For health limitations, however, the risk for poor health peaked at two negative behaviors.

5.2 LIMITATIONS OF THIS RESEARCH

This research is limited for several reasons because the data do not contain pertinent information for fully understanding child health. This is particularly the case in the investigation of child asthma. Gestational age is an important predictor of child asthma because it is an excellent measure developed respiratory function development (Bernsen et al 2005, Gorman & Landale 2005, Yuan et al 2002), but is not available in the NHIS data. In addition, there is no information on the type and frequency of child exposure to allergens in the home, at school, or in daycare. This is important information

for determining the risks for both asthma and ear infections (Arif et al 2004, Celedón et al 2004, von Maffei et al 2001). Information on whether the child was breastfed is also absent. Breastfeeding has been demonstrated to be protective against asthma and ear infections (Oddy & Peat 2003, Oddy et al 1999, Duffy et al 1997, Duncan et al 1993). In addition, there is only data available for one parent in the household. This limits the extent to which the parental health behaviors and history of asthma can truly predict poor health. The responding parent may have better or worse health behaviors than the other parent in the home.

Another limitation is the absence of rural and urban location. These data were available for some of the years selected for this research, but not all. This information would have been particularly salient for AI/AN children. According to 2000 Census reports, 42.6% of all AI/ANs are rural residents (Hobbs & Stoops 2002). The data on child injury are likely biased as NHIS suggests injuries are underreported. Thus, the results on this outcome must be reviewed with caution, especially given the small population size of AI/ANs. It is also difficult to interpret the results for health limitation, as this condition may refer to any number of health problems. There is little research on health limitation as a global measure of child health. Finally, as noted throughout this report, the small N resulted in small cell sizes for AI/ANs prevented either meaningful or stable estimates in many cases (see Appendix A-1).

5.3 CONCLUSION

Aside from listing the specific aims, chapter one also provided a theoretical framework for this project. The theory that best fits this research was one which argues health disparities are the results of the complex associations between biology, social and physical environments, and health behaviors (DHHS 2000). This framework provided a useful approach to understanding AI/AN child health. This was particularly evident in

the race-inclusive models, where consistent measures of child characteristics, SES, and parental health were compared across race-ethnic groups. It was also useful to analyze AI/AN children in aggregate, rather than at the tribal-level, allowing for generalization to the AI/AN population (Barnes et al 2005). While public health policy and initiatives may be better implemented at the tribal-level, a general understanding of AI/AN child health is also an important step in designing policy for this group.

As with any population-based data, the N for AI/ANs is very small. Future projects that intend data collection of AI/ANs should consider oversampling this population to avoid small cell sizes. The Early Childhood Longitudinal Study – Birth Cohort (ECLS–B) oversampled AI/AN infants in 2001 with success. To do so, they selected an additional 18 primary sampling units (PSUs) where the population reported a higher proportion of AI/AN births (Flanagan & Park 2005). This is a challenging prospect, however, as the Internal Review Board for any tribe that falls into such a PSU must grant permission for data collection. In the case of ECLS–B, the Navajo Nation did not approve the IRB application, and thus no data were collected from these reservations.

Another alternative to improving research on AI/ANs would be to compile a data set using IHS clinic records and possibly even discharge records from hospitals that serve IHS areas (there are 12 major areas). Currently, the data available from IHS is in the form of reports and publications. No raw data is publicly available. The IHS clinic records would advise the frequency and nature of visits to the clinic. The hospital discharge records would advise on a number of issues related to hospital care, included diagnoses, procedures, and length of stay. It should be possible to compile health-related data using these sources and still maintain confidentiality of the participants.

Another issue that requires improvement is the measurement of SES for AI/ANs, specifically the measure of insurance status. For the most part, government insurance is

considered an indicator of low SES. The Indian Health Service, however, is not insurance. It is a series of tribal-local clinics that serve federally recognized AI/ANs. Thus, AI/ANs with and without insurance can access this service. As a results IHS offsets access to care for those AI/ANs who do not have insurance. Zuckerman et al (2004) found that AI/ANs with only IHS coverage had similar utilization patterns compared to insured Whites. Over 86% of children in the NHIS data (1991-2003 weighted) who reported IHS as their health care provider had visited a health professional in the last year. They also found that AI/ANs with only IHS coverage fared better than uninsured AI/ANs as well as uninsured Whites for key measures, but received less preventative care. It is difficult to know how to compare this important component of AI/AN health to other race-ethnic groups, because other groups do not have access to such a service. This research included information on the IHS as a form of government insurance in the race-inclusive models, for lack of a better approach. In the race-specific models, IHS was listed separately for AI/ANs. Use of the IHS protected against asthma, health limitation, and injury in the race-specific models, though the ORs were not significant. Interestingly, the OR for IHS in the model for ear infections was significant, but was a risk for recurrent ear infections.

Overall, this research demonstrated that AI/AN children are at greater risk for asthma and recurrent ear infections compared to the other identifiable race-ethnic groups included in this research. While the IHS may mediate this risk, other public health precautions are needed to prevent these illnesses in AI/AN children. Although this group is a very small proportion of the U.S. population, the health of AI/ANs is demonstrative of the long-term commitment the U.S. government made to AI/AN tribes.[13] The health

[13] Federally recognized American Indian Tribes and Alaska Native corporations enjoy a government-to-government relationship with the United States of America. This unique relationship has been given substance through numerous Supreme Court decisions, treaties, legislation, and Executive Orders (IHS 2005).

of AI/ANs has improved over time, with lower mortality attributable to infectious disease (Young 1997). However, as this research demonstrates, there is room for much needed progress.

Appendix A. Weighted Distribution of Variables Included in Logistic Regression Models by Race-Ethnicity.

Variable Categories	Percent Distribution						
	Non-Hisp AI/AN	Non-Hisp White	Non-Hisp Black	Mexican Origin	Non-Hisp Asian	Non-Hisp Other	Other Hispanic
CHILD CHARACTERISTICS							
Sex							
Male	54.60	51.32	50.52	50.81	51.09	50.58	51.49
Female	45.40	48.68	49.48	49.19	48.91	49.42	48.51
Age							
0-3	20.50	20.95	20.51	25.01	23.07	25.34	23.21
4-7	22.30	21.96	22.20	24.57	21.68	28.67	24.13
8-11	21.57	23.56	24.22	23.02	24.16	22.48	23.33
12-17	35.63	33.52	33.07	27.41	31.09	23.51	29.33
Birthweight (grams)							
Very low (< 1500)	2.90	0.82	2.07	1.04	1.05	1.20	1.17
Low (1500-2499)	3.27	4.82	8.99	5.58	7.55	5.75	5.92
Normal (2500-3999)	79.32	79.38	80.94	81.23	86.84	80.63	81.23
High (4000 +)	14.51	14.99	8.01	12.16	4.56	12.41	11.68
SOCIODEMOGRAPHIC CHARACTERISTICS							
Family Income to Poverty Ratio							
Poor (< 1.00)	32.63	9.73	35.16	35.98	17.31	20.83	29.65
Near Poor (1.00-1.99)	34.43	17.11	25.16	32.83	16.01	19.52	27.79
Not Poor (> 2.00)	32.94	73.15	39.68	31.19	66.68	59.65	42.56
Parental Educational Attainment							
No high school diploma	24.45	8.70	19.83	53.51	14.06	10.67	31.15
High school diploma or GED	32.96	28.71	33.34	21.89	17.22	26.67	27.15
Some college	32.32	32.00	34.35	18.47	21.28	35.97	27.45
College degree	10.27	30.59	12.48	6.13	47.44	26.70	14.26

Appendix A. Continued.

Variable Categories	Percent Distribution						
	Non-Hisp AI/AN	Non-Hisp White	Non-Hisp Black	Mexican Origin	Non-Hisp Asian	Non-Hisp Other	Other Hispanic
Parent Household Composition							
One parent in household	36.98	16.81	56.56	21.77	12.91	29.60	32.78
Both parents in household	63.02	83.19	43.44	78.23	87.09	70.40	67.22
Insurance Status							
No insurance	3.55	4.93	7.13	18.48	9.46	6.13	11.11
Government insurance	47.82	11.00	33.69	30.40	16.15	25.89	32.02
Private insurance	48.63	84.07	59.18	51.12	74.39	67.98	56.86
ENVIRONMENTAL FACTORS							
Parent Use of Alcohol							
Does not drink	47.22	27.86	48.06	47.80	59.42	33.70	44.50
Light drinker	44.46	62.65	45.50	45.23	36.29	58.38	49.72
Moderate or heavy drinker	4.29	9.49	8.32	6.97	7.91	6.97	5.78
Parent Smoking Status							
Never smoked	45.29	54.31	66.53	71.44	77.27	54.12	65.46
Former smoker	16.22	19.50	9.88	12.41	10.37	18.54	13.45
Light smoker	13.79	5.15	9.77	9.88	5.69	6.03	10.06
Heavy smoker	20.35	16.61	12.53	5.64	6.30	18.11	9.69
Heavy plus smoker	4.36	4.43	1.30	0.63	0.37	3.20	1.33
Parent Self-rated Health							
Excellent or very good	59.00	76.19	61.75	61.24	70.28	66.90	64.56
Good	30.06	18.70	26.97	29.74	23.85	24.32	25.23
Fair or poor	10.94	5.11	11.28	9.02	5.87	8.78	10.21

Appendix A. Continued.

Variable Categories	Percent Distribution						
	Non-Hisp AI/AN	Non-Hisp White	Non-Hisp Black	Mexican Origin	Non-Hisp Asian	Non-Hisp Other	Other Hispanic
Parent Weight Status (BMI)							
Underweight (< 18.5)	1.37	2.11	1.21	0.85	3.51	1.27	1.36
Normal (18.5-24.9)	27.94	42.06	27.87	29.19	54.93	40.43	38.17
Overweight (25.0-29.9)	33.41	32.92	33.36	38.59	28.13	28.64	34.41
Obese (30.0-39.9)	29.10	17.37	28.10	23.24	8.49	22.45	20.18
Morbidly obese (40.0 +)	5.47	2.00	5.86	2.61	0.36	3.46	2.26
Weight unknown	2.71	3.53	3.59	5.52	4.58	3.75	3.61
Cumulation of Parent Health Behaviors							
No negative behaviors	14.59	28.87	20.07	24.05	50.16	28.39	30.18
One negative behavior	56.74	52.65	60.96	60.58	41.92	51.72	53.86
Two negative behaviors	25.25	16.58	16.93	13.61	7.48	17.83	14.42
Three negative behavrios	3.42	1.90	2.04	1.76	0.44	2.06	1.54
Genetic Proxy for Asthma							
Parent has asthma	13.34	9.90	10.93	5.70	5.80	14.52	11.97
Parent does not have asthma	86.66	90.10	89.07	94.30	94.20	85.48	88.03
Outcome Variables							
Asthma	17.80	11.38	15.78	8.17	10.21	17.06	15.04
3+ ear infections	8.98	7.21	5.13	6.53	3.04	6.56	6.90
Health limitation	3.40	2.14	2.32	1.61	1.03	2.96	2.37
Injury	3.43	3.31	1.64	1.33	0.47	2.34	2.15

Appendix A-I. Unweighted Frequencies for Variables Included in Logistic Regression Models for AI/ANs (unweighted n = 371).

Variable Categories	Unweighted Frequency
CHILD CHARACTERISTICS	
Sex	
Male	204
Female	167
Age	
0-3	87
4-7	81
8-11	73
12-17	130
Birthweight (grams)	
Very low (< 1500)	9
Low (1500-2499)	16
Normal (2500-3999)	286
High (4000 +)	60
SOCIODEMOGRAPHIC CHARACTERISTICS	
Family Income to Poverty Ratio	
Poor (< 1.00)	114
Near Poor (1.00-1.99)	111
Not Poor (> 2.00)	146
Parental Educational Attainment	
No high school diploma	84
High school diploma or GED	125
Some college	115
College degree	47
Parent Household Composition	
One parent in household	154
Both parents in household	217
Insurance Status	
No insurance	12
Government insurance	86
Private insurance	141
Indian Health Service	132
ENVIRONMENTAL FACTORS	
Parent Use of Alcohol	
Does not drink	172
Light drinker	168
Moderate or heavy drinker	31

Appendix A-I Continued.

Parent Smoking Status	
Never smoked	169
Former smoker	54
Light smoker	46
Heavy smoker	82
Heavy plus smoker	20
Parent Self-rated Health	
Excellent or very good	220
Good	100
Fair or poor	51
Parent Weight Status (BMI)	
Underweight (< 18.5)	7
Normal (18.5-24.9)	100
Overweight (25.0-29.9)	128
Obese (30.0-39.9)	107
Morbidly obese (40.0 +)	19
Weight unknown	10
Cumulation of Parent Health Behaviors	
No negative behaviors	59
One negative behavior	193
Two negative behaviors	107
Three negative behavrios	12
Genetic Proxy for Asthma	
Parent has asthma	54
Parent does not have asthma	317
Outcome Variables	
Asthma	68
3+ ear infections	34
Health limitation	17
Injury	15

Appendix B. Logistic Regression Estimates (Odds Ratios) for Child Having Asthma,† NHIS 1997-2003 Weighted.¶

Variables		Baseline Race Only	Model 2 (+) Child Characteristics	Model 3 (+) Family / Parent Characteristics	Full Model (+) Genetic Proxy	ALT Full Model (Δ) Parental Health Behaviors
	Constant	-2.05 ***	-1.65 ***	-1.73 ***	-1.78 ***	-1.75 ***
CHILD CHARACTERISTICS						
Race/Ethnicity	Non-Hispanic White	**1.00**	**1.00**	**1.00**	**1.00**	**1.00**
	Non-Hispanic AI/AN	1.69 *	1.66 *	1.37 ++	1.37 ++	1.36 ++
	Non-Hispanic Black	1.46 ***	1.43 ***	1.21 ***	1.24 ***	1.22 ***
	Mexican Origin	0.69 ***	0.71 ***	0.71 ***	0.76 ***	0.74 ***
	Non-Hispanic Asian	0.89	0.89	0.96	1.02	0.98
	Non-Hispanic Other	1.60 ***	1.69 ***	1.53 ***	1.48 **	1.47 **
	Other Hispanic	1.38 ***	1.41 ***	1.32 ***	1.31 ***	1.29 ***
Sex	Male		**1.00**	**1.00**	**1.00**	**1.00**
	Female		0.66 ***	0.66 ***	0.65 ***	0.65 ***
Age	0-3		0.38 ***	0.41 ***	0.40 ***	0.40 ***
	4-7		0.77 ***	0.81 ***	0.80 ***	0.80 ***
	8-11		0.87 ***	0.91 *	0.90 *	0.90 *
	12-17		**1.00**	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)		2.35 ***	2.20 ***	2.22 ***	2.22 ***
	Low (1500-2499)		1.48 ***	1.43 ***	1.42 ***	1.42 ***
	Normal (2500-3999)		**1.00**	**1.00**	**1.00**	**1.00**
	High (4000+)		0.99	0.99	1.00	1.00
FAMILY / PARENT CHARACTERISTICS						
Socioeconomic Factors						
Family Income to Poverty Ratio	Poor (< 1.00)			1.06	1.04	1.04
	Near Poor (1.00 - 1.99)			0.94 ++	0.93 ++	0.93 ++
	Not Poor (2.00 +)			**1.00**	**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma			0.84 **	0.86 *	0.86 *
	High School Diploma or GED			0.91 *	0.93 +++	0.93 ++
	Some College			**1.00**	**1.00**	**1.00**
	College Degree			0.91 +++	0.91 +++	0.90 *
Parent Household Composition	One Parent in Household			1.25 ***	1.21 ***	1.22 ***
	Both Parents in Household			**1.00**	**1.00**	**1.00**
Source of Insurance‡	No Insurance			0.86 *	0.87 +++	0.87 +++
	Government Insurance			1.12 *	1.11 *	1.12 *
	Private Insurance			**1.00**	**1.00**	**1.00**
Environmental Factors						
Parent Use of Alcohol	Does Not Drink			0.95	0.97	…
	Light Drinker			**1.00**	**1.00**	…
	Moderate to Heavy Drinker			0.98	0.99	…
Parent Smoking Status	Never Smoked			**1.00**	**1.00**	…
	Former Smoker			1.17 **	1.15 **	…
	Light Smoker			1.18 *	1.17 *	…
	Heavy Smoker			1.21 ***	1.20 ***	…
	Heavy + Smoker			1.26 *	1.25 *	…
Parent Self-Rated Health	Excellent or Very Good			0.77 ***	0.82 ***	0.81 ***
	Good			**1.00**	**1.00**	**1.00**
	Fair or Poor			1.26 ***	1.13 +++	1.14 +++
Parent Weight Status	Underweight			1.07	1.09	…
	Normal			**1.00**	**1.00**	…
	Overweight			1.11 *	1.10 *	…
	Obese			1.22 ***	1.18 ***	…
	Morbidly Obese			1.31 **	1.19 +++	…
	Weight Unknown			0.97	0.97	…
Genetic Proxy	Parent Asthma				2.87 ***	2.89 ***
	No Parent Asthma				**1.00**	**1.00**
Accumulation of Parental Health Behaviors§	No Negative Outcomes					**1.00**
	One Negative Outcome					1.13 **
	Two Negative Outcomes					1.26 ***
	Three Negative Outcomes					1.28 +++
	Pseudo R^2	0.0061	0.0290	0.0395	0.0592	0.0585
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0089	0.0416	0.0562	0.0830	0.0820
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0045	0.0210	0.0286	0.0424	0.0419
	df	6	13	33	35	27
	-2LL	49417	48279	47757	46780	46816
	Unweighted N	67903	67903	67903	67903	67903

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "Has a doctor or health professional ever told you that [child] had asthma?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix B-I. Logistic Regression Estimates (Odds Ratios) for Child Having Asthma,† NHIS 1997-2003 Weighted.¶

Non Hispanic American Indians / Alaska Natives		**Baseline**	**Model 2**	**Full Model**	**ALT Full Model**
Variables		(+) Child Characteristics	(+) Family / Parent Characteristics	(+) Genetic Proxy	(Δ) Parental Health Behaviors
	Constant	-1.20 **	-0.51	-0.96 ++	-1.61 +++
CHILD CHARACTERISTICS					
Sex	Male	**1.00**	**1.00**	**1.00**	**1.00**
	Female	0.55 ++	0.56 ++	0.62	0.59 ++
Age	0-4	0.57	0.64	0.68	0.68
	5-11	0.98	1.03	1.10	1.16
	12-17	**1.00**	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	1.45	1.04	1.31	1.32
	Low (1500-2499)	2.20	2.05	1.62	1.66
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**	**1.00**
	High (3500+)	1.02	0.91	0.98	0.97
FAMILY / PARENT CHARACTERISTICS					
Socioeconomic Factors					
Family Income to Poverty Ratio	Poor (< 1.00)		0.76	0.77	0.78
	Not Poor (1.00+)		**1.00**	**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.59	0.69	0.69
	High School Diploma or GED		**1.00**	**1.00**	**1.00**
	At Least Some College		0.55 ++	0.51 ++	0.51 ++
Parent Household Composition	One Parent in Household		2.00 ++	2.23 +++	2.06 ++
	Both Parents in Household		**1.00**	**1.00**	**1.00**
Source of Insurance‡	Government Insurance		0.70	0.72	0.69
	Indian Health Service		0.42 ++	0.46	0.54 ++
	Private Insurance		**1.00**	**1.00**	**1.00**
Environmental Factors					
Parent Smoking Status	Never Smoked		**1.00**	**1.00**	…
	Former / Current Smoker		0.57 ++	0.55 ++	…
Parent Self-Rated Health	Excellent or Very Good		1.04	1.15	1.22
	Good		**1.00**	**1.00**	**1.00**
	Fair or Poor		1.06	0.94	1.30
Parental Weight Status	Underweight		0.00 ***	0.00 ***	…
	Normal		**1.00**	**1.00**	…
	Overweight		0.78	0.74	…
	Obese		1.91 ++	1.78	…
	Weight Unknown		0.95	0.84	…
Genetic Proxy	Parent Asthma			4.23 **	4.32 **
	No Parent Asthma			**1.00**	**1.00**
Accumulation of Parental Health Behaviors§	No Negative Outcomes				**1.00**
	1 Negative Outcome				1.86
	2 to 3 Negative Outcomes				1.00
	Pseudo R^2	0.0228	0.1170	0.1597	0.1191
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0418	0.1968	0.2585	0.1999
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0211	0.1038	0.1389	0.1055
	df	6	19	20	17
	-2LL	340	307	292	306
	Unweighted N	371	371	371	371

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "Has a doctor or health professional ever told you that [child] had asthma?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix B-II. Logistic Regression Estimates (Odds Ratios) for Child Having Asthma[†], NHIS 1997-2003 Weighted.[¶]

Non Hispanic Whites		**Baseline**	**Model 2**	**Full Model**	**ALT Full Model**
Variables		(+) Child Characteristics	(+) Family / Parent Characteristics	(+) Genetic Proxy	(Δ) Parental Health Behaviors
	Constant	-1.55 ***	-1.67 ***	-1.80 ***	-1.72 ***
CHILD CHARACTERISTICS					
Sex	Male	**1.00**	**1.00**	**1.00**	**1.00**
	Female	0.66 ***	0.66 ***	0.66 ***	0.65 ***
Age	0-4	0.37 ***	0.39 ***	0.38 ***	0.38 ***
	5-11	0.79 ***	0.82 ***	0.81 ***	0.80 ***
	12-17	**1.00**	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	1.97 ***	1.29	1.28	1.76 **
	Low (1500-2499)	1.42 ***	1.38 ***	1.37 **	1.37 ***
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**	**1.00**
	High (3500+)	0.91 **	0.93 +++	0.93 ++	0.93 ++
FAMILY / PARENT CHARACTERISTICS					
Socioeconomic Factors					
Family Income to Poverty Ratio	Poor (< 1.00)		1.16 +++	1.14 ++	1.14 ++
	Not Poor (1.00+)		**1.00**	**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.99	0.97	0.97
	High School Diploma or GED		**1.00**	**1.00**	**1.00**
	At Least Some College		1.06	1.03	1.02
Parent Household Composition	One Parent in Household		1.16 *	1.14 *	1.14 *
	Both Parents in Household		**1.00**	**1.00**	**1.00**
Source of Insurance[‡]	No Insurance		0.97	0.98	0.98
	Government Insurance		1.13 ++	1.12 ++	1.12 ++
	Private Insurance		**1.00**	**1.00**	**1.00**
Environmental Factors					
Parent Smoking Status	Never Smoked		**1.00**	**1.00**	…
	Former / Current Smoker		1.18 ***	1.17 ***	…
Parent Self-Rated Health	Excellent or Very Good		0.77 ***	0.82 ***	0.81 ***
	Good		**1.00**	**1.00**	**1.00**
	Fair or Poor		1.37 ***	1.24 *	1.24 *
Parental Weight Status	Underweight		1.12	1.13	…
	Normal		**1.00**	**1.00**	…
	Overweight		1.13 *	1.11 *	…
	Obese		1.21 ***	1.16 *	…
	Weight Unknown		1.02	1.02	…
Genetic Proxy	Parent Asthma			2.89 ***	2.91 ***
	No Parent Asthma			**1.00**	**1.00**
Accumulation of Parental Health Behaviors[§]	No Negative Outcomes				**1.00**
	1 Negative Outcome				1.09 ++
	2 to 3 Negative Outcomes				1.21 **
	Pseudo R^2	0.0251	0.0348	0.0549	0.0542
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0350	0.0482	0.0749	0.0740
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0177	0.0244	0.0382	0.0377
	df	6	19	20	17
	-2LL	26118	25858	25320	25339
	Unweighted N	37,789	37,789	37,789	37,789

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "Has a doctor or health professional ever told you that [child] had asthma?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix B-III. Logistic Regression Estimates (Odds Ratios) for Child Having Asthma,† NHIS 1997-2003 Weighted.¶

Non Hispanic Blacks		**Baseline**	**Model 2**	**Full Model**	**ALT Full Model**
Variables		(+) Child Characteristics	(+) Family / Parent Characteristics	(+) Genetic Proxy	(Δ) Parental Health Behaviors
	Constant	-1.59 ***	-1.82 ***	-1.88 ***	-1.94 ***
CHILD CHARACTERISTICS					
Sex	Male	**1.00**	**1.00**	**1.00**	**1.00**
	Female	0.71 ***	0.70 ***	0.70 ***	0.70 ***
Age	0-4	0.76 *	0.81 +++	0.80 +++	0.79 +++
	5-11	1.11	1.15 ++	1.14 ++	1.14 ++
	12-17	**1.00**	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	2.44 ***	1.60 +++	1.62 +++	2.50 ***
	Low (1500-2499)	1.58 ***	1.53 ***	1.55 ***	1.55 ***
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**	**1.00**
	High (3500+)	1.08	1.07	1.07	1.07
FAMILY / PARENT CHARACTERISTICS					
Socioeconomic Factors					
Family Income to Poverty Ratio	Poor (< 1.00)		1.01	1.00	1.00
	Not Poor (1.00+)		**1.00**	**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		1.02	1.02	1.01
	High School Diploma or GED		**1.00**	**1.00**	**1.00**
	At Least Some College		1.06	1.04	1.04
Parent Household Composition	One Parent in Household		1.15 ++	1.13 ++	1.12 ++
	Both Parents in Household		**1.00**	**1.00**	**1.00**
Source of Insurance‡	No Insurance		1.05	1.04	1.04
	Government Insurance		1.18 ++	1.18 ++	1.18 ++
	Private Insurance		**1.00**	**1.00**	**1.00**
Environmental Factors					
Parent Smoking Status	Never Smoked		**1.00**	**1.00**	…
	Former / Current Smoker		1.22 **	1.21 *	…
Parent Self-Rated Health	Excellent or Very Good		0.77 **	0.80 *	0.79 +++
	Good		**1.00**	**1.00**	**1.00**
	Fair or Poor		1.15	1.06	1.06
Parental Weight Status	Underweight		0.69	0.73	…
	Normal		**1.00**	**1.00**	…
	Overweight		1.10	1.10	…
	Obese		1.23 +++	1.20 ++	…
	Weight Unknown		0.82	0.82	…
Genetic Proxy	Parent Asthma			2.05 ***	2.08 ***
	No Parent Asthma			**1.00**	**1.00**
Accumulation of Parental Health Behaviors§	No Negative Outcomes				**1.00**
	1 Negative Outcome				1.29 *
	2 to 3 Negative Outcomes				1.40 *
	Pseudo R^2	0.0123	0.0231	0.0325	0.0317
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0212	0.0395	0.0550	0.0537
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0107	0.0200	0.0279	0.0272
	df	6	19	20	17
	-2LL	8240	8150	8072	8078
	Unweighted N	9,570	9,570	9,570	9,570

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "Has a doctor or health professional ever told you that [child] had asthma?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix B-IV. Logistic Regression Estimates (Odds Ratios) for Child Having Asthma,† NHIS 1997-2003 Weighted.¶

Mexican Origin Variables		**Baseline** (+) Child Characteristics	**Model 2** (+) Family / Parent Characteristics	**Full Model** (+) Genetic Proxy	**ALT Full Model** (Δ) Parental Health Behaviors
	Constant	-2.00 ***	-2.06 ***	-2.15 ***	-2.24 ***
CHILD CHARACTERISTICS					
Sex	Male	**1.00**	**1.00**	**1.00**	**1.00**
	Female	0.63 ***	0.62 ***	0.61 ***	0.61 ***
Age	0-4	0.46 ***	0.50 ***	0.50 ***	0.49 ***
	5-11	0.79 +++	0.82 +++	0.83 ++	0.83 ++
	12-17	**1.00**	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	2.68 **	1.98 +++	2.06 +++	2.52 *
	Low (1500-2499)	1.30 ++	1.26	1.25	1.25
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**	**1.00**
	High (3500+)	1.17 ++	1.11	1.09	1.10
FAMILY / PARENT CHARACTERISTICS					
Socioeconomic Factors					
Family Income to Poverty Ratio	Poor (< 1.00)		0.89	0.87	0.86
	Not Poor (1.00+)		**1.00**	**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.79 +++	0.82 ++	0.80 +++
	High School Diploma or GED		**1.00**	**1.00**	**1.00**
	At Least Some College		1.36 *	1.32 *	1.33 *
Parent Household Composition	One Parent in Household		1.43 **	1.36 **	1.37 ***
	Both Parents in Household		**1.00**	**1.00**	**1.00**
Source of Insurance‡	No Insurance		0.71 +++	0.75 +++	0.74 +++
	Government Insurance		1.04	1.06	1.06
	Private Insurance		**1.00**	**1.00**	**1.00**
Environmental Factors					
Parent Smoking Status	Never Smoked		**1.00**	**1.00**	…
	Former / Current Smoker		1.16 ++	1.32	…
Parent Self-Rated Health	Excellent or Very Good		0.84 +++	0.85 ++	0.85 ++
	Good		**1.00**	**1.00**	**1.00**
	Fair or Poor		1.20	1.10	1.12
Parental Weight Status	Underweight		1.00	1.04	…
	Normal		**1.00**	**1.00**	…
	Overweight		1.14	1.14	…
	Obese		1.34 *	1.29 +++	…
	Weight Unknown		0.67 ++	0.67 ++	…
Genetic Proxy	Parent Asthma			3.08 ***	3.11 ***
	No Parent Asthma			**1.00**	**1.00**
Accumulation of Parental Health Behaviors§	No Negative Outcomes				**1.00**
	1 Negative Outcome				1.34 *
	2 to 3 Negative Outcomes				1.39 +++
	Pseudo R^2	0.0207	0.0417	0.0570	0.0561
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0232	0.0461	0.0624	0.0615
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0211	0.1038	0.1389	0.1055
	df	6	19	20	17
	-2LL	5946	5819	5726	5732
	Unweighted N	10,732	10,732	10,732	10,732

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "Has a doctor or health professional ever told you that [child] had asthma?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix C. Logistic Regression Estimates (Odds Ratios) for Child Having 3+ Ear Infections in the Last Year,† NHIS 1997-2003 Weighted.¶

Variables		Baseline Race Only	Model 2 (+) Child Characteristics	Full Model (+) Family / Parent Characteristics	ALT Full Model (Δ) Parental Health Behaviors
	Constant	-2.55 ***	-3.46 ***	-3.47 ***	-3.51 ***
CHILD CHARACTERISTICS					
Race/Ethnicity	Non-Hispanic White	**1.00**	**1.00**	**1.00**	**1.00**
	Non-Hispanic AI/AN	1.27	1.26	1.07	1.05
	Non-Hispanic Black	0.70 ***	0.68 ***	0.58 ***	0.56 ***
	Mexican Origin	0.90 +++	0.82 ***	0.82 +++	0.79 ***
	Non-Hispanic Asian	0.40 ***	0.38 ***	0.42 ***	0.40 ***
	Non-Hispanic Other	0.90	0.79	0.72 *	0.71 *
	Other Hispanic	0.95	0.89 ++	0.85 +++	0.82 *
Sex	Male		**1.00**	**1.00**	**1.00**
	Female		0.94 ++	0.95 ++	0.95 ++
Age	0-3		5.29 ***	5.76 ***	5.74 ***
	4-7		3.30 ***	3.48 ***	3.49 ***
	8-11		1.67 ***	1.73 ***	1.74 ***
	12-17		**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)		1.92 ***	1.82 ***	1.80 *
	Low (1500-2499)		1.25 *	1.19 +++	1.20 +++
	Normal (2500-3999)		**1.00**	**1.00**	**1.00**
	High (4000+)		1.01	1.01	1.01
FAMILY / PARENT CHARACTERISTICS					
Socioeconomic Factors					
Family Income to Poverty Ratio	Poor (< 1.00)			0.99	0.97
	Near Poor (1.00 - 1.99)			0.96	0.94
	Not Poor (2.00 +)			**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma			0.91 ++	0.90 ++
	High School Diploma or GED			1.01	1.01
	Some College			**1.00**	**1.00**
	College Degree			0.99	0.98
Parent Household Composition	One Parent in Household			1.22 ***	1.24 ***
	Both Parents in Household			**1.00**	**1.00**
Source of Insurance‡	No Insurance			0.86 +++	0.85 +++
	Government Insurance			1.22 **	1.22 **
	Private Insurance			**1.00**	**1.00**
Environmental Factors					
Parent Use of Alcohol	Does Not Drink			0.86 **	…
	Light Drinker			**1.00**	…
	Moderate to Heavy Drinker			1.05	…
Parent Smoking Status	Never Smoked			**1.00**	…
	Former Smoker			1.07	…
	Light Smoker			1.07	…
	Heavy Smoker			1.21 **	…
	Heavy + Smoker			1.30 *	…
Parent Self-Rated Health	Excellent or Very Good			0.78 ***	0.78 ***
	Good			**1.00**	**1.00**
	Fair or Poor			1.38 ***	1.38 ***
Parent Weight Status	Underweight			1.01	…
	Normal			**1.00**	…
	Overweight			1.09 +++	…
	Obese			1.17 *	…
	Morbidly Obese			1.28 +++	…
	Weight Unknown			0.96	…
Accumulation of Parental Health Behaviors§	No Negative Outcomes				**1.00**
	One Negative Outcome				1.18 **
	Two Negative Outcomes				1.30 ***
	Three Negative Outcomes				1.41 *
	Pseudo R^2	0.0034	0.0544	0.0636	0.0627
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0034	0.0522	0.0609	0.0600
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0017	0.0265	0.0309	0.0304
	df	6	13	34	26
	-2LL	33384	31677	31367	31399
	Unweighted N	67903	67903	67903	67903

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "During the past 12 months, has [child] had three or more ear infections?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix C-I. Logistic Regression Estimates (Odds Ratios) for Child Having 3+ Ear Infections in the Last Year,† NHIS 1997-2003 Weighted.¶

Non Hispanic American Indians / Alaska Natives		**Baseline**	**Full Model**	**ALT Full Model**
Variables		(+) Child Characteristics	(+) Family / Parent Characteristics	(Δ) Parental Health Behaviors
	Constant	-2.46 ***	-2.58 ***	-3.38 **
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	0.77	0.83	0.86
Age	0-4	0.85	1.04	1.08
	5-11	1.23	1.19	1.18
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	0.85	0.52	0.53
	Low (1500-2499)	1.26	1.61	1.48
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	1.54	1.36	1.34
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		0.30 +++	0.29 +++
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.74	0.73
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		1.31	1.35
Parent Household Composition	One Parent in Household		1.51	1.53
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance‡	Government Insurance		1.21	1.28
	Indian Health Service		2.46 +++	2.59 +++
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Smoking Status	Never Smoked		**1.00**	…
	Former / Current Smoker		1.73	…
Parent Weight Status	Underweight		0.00	…
	Normal		**1.00**	…
	Overweight		0.84 ***	…
	Obese		0.89	…
	Unknown		0.48	…
Parent Self-Rated Health	Excellent or Very Good		0.44 ++	0.48 ++
	Good		**1.00**	**1.00**
	Fair or Poor		0.42	0.44
Cumulation of Parental Health Behaviors§	No Negative Outcomes			**1.00**
	1 Negative Outcome			2.56
	2 to 3 Negative Outcomes			3.01
	Pseudo R^2	0.0126	0.1450	0.1018
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0151	0.1608	0.1157
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0076	0.0839	0.0596
	df	6	19	16
	-2LL	221	192	201
	Unweighted N	371	371	371

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "During the past 12 months, has [child] had three or more ear infections?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix C-II. Logistic Regression Estimates (Odds Ratios) for Child Having 3+ Ear Infections in the Last Year,† NHIS 1997-2003 Weighted.¶

Non Hispanic Whites		**Baseline**	**Full Model**	**ALT Full Model**
Variables		(+) Child Characteristics	(+) Family / Parent Characteristics	(Δ) Parental Health Behaviors
	Constant	-3.52 ***	-3.56 ***	-3.68 ***
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	0.96	0.97	0.97
Age	0-4	5.42 ***	5.74 ***	5.73 ***
	5-11	2.33 ***	2.42 ***	2.41 ***
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	2.41 ***	2.24 ***	2.23 ***
	Low (1500-2499)	1.36 *	1.32 *	1.32 *
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	0.99	1.01	1.01
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		0.99	0.99
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.99	0.99
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		1.00	1.01
Parent Household Composition	One Parent in Household		1.18 *	1.18 *
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance‡	No Insurance		0.83 ++	0.83 ++
	Government Insurance		1.30 **	1.30 **
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Smoking Status	Never Smoked		**1.00**	…
	Former / Current Smoker		1.14 *	…
Parent Weight Status	Underweight		1.04	…
	Normal		**1.00**	…
	Overweight		1.15 *	…
	Obese		1.16 +++	…
	Unknown		0.96	…
Parent Self-Rated Health	Excellent or Very Good		0.85 *	0.85 *
	Good		**1.00**	**1.00**
	Fair or Poor		1.38 **	1.39 **
Cumulation of Parental Health Behaviors§	No Negative Outcomes			**1.00**
	1 Negative Outcome			1.22 **
	2 to 3 Negative Outcomes			1.34 ***
	Pseudo R^2	0.0527	0.0594	0.0597
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0532	0.0597	0.0600
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0269	0.0303	0.0303
	df	6	19	16
	-2LL	18553	18421	18417
	Unweighted N	37,789	37,789	37,789

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "During the past 12 months, has [child] had three or more ear infections?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix C-III. Logistic Regression Estimates (Odds Ratios) for Child Having 3+ Ear Infections in the Last Year,† NHIS 1997-2003 Weighted.¶

Non Hispanic Blacks		**Baseline**	**Full Model**	**ALT Full Model**
Variables		(+) Child Characteristics	(+) Family / Parent Characteristics	(Δ) Parental Health Behaviors
	Constant	-3.75 ***	-3.89 ***	-3.89 ***
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	0.78 +++	0.77 +++	0.77 +++
Age	0-4	4.78 ***	5.21 ***	5.20 ***
	5-11	1.91 ***	2.02 ***	2.01 ***
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	1.66 ++	1.66 ++	1.65 ++
	Low (1500-2499)	1.26 +	1.18	1.18
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	1.26 ++	1.25 ++	1.26 ++
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		1.06	1.06
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.79	0.78 ++
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		0.97	0.97
Parent Household Composition	One Parent in Household		1.30 ++	1.30 ++
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance‡	No Insurance		0.60 +++	0.59 +++
	Government Insurance		1.24 ++	1.23 ++
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Smoking Status	Never Smoked		**1.00**	…
	Former / Current Smoker		1.12	…
Parent Weight Status	Underweight		1.06	…
	Normal		**1.00**	…
	Overweight		1.16	…
	Obese		1.24	…
	Unknown		1.13	…
Parent Self-Rated Health	Excellent or Very Good		0.62 ***	0.62 **
	Good		**1.00**	**1.00**
	Fair or Poor		1.21	1.21
Cumulation of Parental Health Behaviors§	No Negative Outcomes			**1.00**
	1 Negative Outcome			1.19 +
	2 to 3 Negative Outcomes			1.42 ++
	Pseudo R^2	0.0487	0.0682	0.0685
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0387	0.0537	0.0539
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0195	0.0272	0.0273
	df	6	16	16
	-2LL	3684	3608	3607
	Unweighted N	9,570	9,570	9,570

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "During the past 12 months, has [child] had three or more ear infections?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix C-IV. Logistic Regression Estimates (Odds Ratios) for Child Having 3+ Ear Infections in the Last Year,† NHIS 1997-2003 Weighted.¶

Mexican Origin

Variables		Baseline (+) Child Characteristics	Full Model (+) Family / Parent Characteristics	ALT Full Model (Δ) Parental Health Behaviors
	Constant	-3.60 ***	-3.35 ***	-3.32 ***
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	1.15 ++	1.15 ++	1.15 ++
Age	0-4	3.98 ***	4.22 ***	4.18 ***
	5-11	2.15 ***	2.25 ***	2.24 ***
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	1.33	1.19	1.19
	Low (1500-2499)	1.19	1.12	1.12
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	0.98	0.97	0.98
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		1.13	1.13
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.74 *	0.73 *
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		0.98	0.97
Parent Household Composition	One Parent in Household		0.98	1.00
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance‡	No Insurance		0.98	0.97
	Government Insurance		1.07	1.07
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Smoking Status	Never Smoked		**1.00**	…
	Former / Current Smoker		1.26 +++	…
Parent Weight Status	Underweight		0.92	…
	Normal		**1.00**	…
	Overweight		0.75 *	…
	Obese		1.10	…
	Unknown		0.87	…
Parent Self-Rated Health	Excellent or Very Good		0.69 **	0.68 **
	Good		**1.00**	**1.00**
	Fair or Poor		1.38 +++	1.43 +++
Cumulation of Parental Health Behaviors§	No Negative Outcomes			**1.00**
	1 Negative Outcome			0.93
	2 to 3 Negative Outcomes			1.14
	Pseudo R^2	0.0314	0.0430	0.0430
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0298	0.0407	0.0407
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0150	0.0206	0.0206
	df	6	19	16
	-2LL	5020	4940	4959
	Unweighted N	10,732	10,732	10,732

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "During the past 12 months, has [child] had three or more ear infections?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix D. Logistic Regression Estimates (Odds Ratios) for Child Having a Health Limitation,† NHIS 1997-2003 Weighted.¶

Variables		Baseline Race Only	Model 2 (+) Child Characteristics	Full Model (+) Family / Parent Characteristics	ALT Full Model (Δ) Parental Health Behaviors
	Constant	-3.82 ***	-3.43 ***	-3.21 ***	-3.23 ***
CHILD CHARACTERISTICS					
Race/Ethnicity	Non-Hispanic White	**1.00**	**1.00**	**1.00**	**1.00**
	Non-Hispanic AI/AN	1.61	1.53	0.95	0.95
	Non-Hispanic Black	1.08	0.99	0.67 **	0.68 **
	Mexican Origin	0.75 *	0.77 *	0.64 ***	0.64 ***
	Non-Hispanic Asian	0.47 +++	0.46 +++	0.47 +++	0.46 +++
	Non-Hispanic Other	1.39 ++	1.47 ++	1.12	1.13
	Other Hispanic	1.11	1.11	0.87	0.86
Sex	Male		**1.00**	**1.00**	**1.00**
	Female		0.90 ++	0.90 ++	0.90 ++
Age	0-3		0.38 ***	0.43 ***	0.42 ***
	4-7		0.53 ***	0.57 ***	0.57 ***
	8-11		0.65 ***	0.69 ***	0.69 ***
	12-17		**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)		5.18 ***	4.45 ***	4.36 ***
	Low (1500-2499)		1.95 ***	1.81 ***	1.79 ***
	Normal (2500-3999)		**1.00**	**1.00**	**1.00**
	High (4000+)		0.75 *	0.75 *	0.75 *
FAMILY / PARENT CHARACTERISTICS					
Socioeconomic Factors					
Family Income to Poverty Ratio	Poor (< 1.00)			1.35 *	1.32 *
	Near Poor (1.00 - 1.99)			1.27 *	1.25 *
	Not Poor (2.00 +)			**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma			0.66 ***	0.65 ***
	High School Diploma or GED			0.85 ++	0.85 *
	Some College			**1.00**	**1.00**
	College Degree			0.80 +++	0.80 +++
Parent Household Composition	One Parent in Household			1.13 ++	1.13 ++
	Both Parents in Household			**1.00**	**1.00**
Source of Insurance‡	No Insurance			0.85	0.84 ++
	Government Insurance			1.46 ***	1.46 ***
	Private Insurance			**1.00**	**1.00**
Environmental Factors					
Parent Use of Alcohol	Does Not Drink			0.93	…
	Light Drinker			**1.00**	…
	Moderate to Heavy Drinker			1.18	…
Parent Smoking Status	Never Smoked			**1.00**	…
	Former Smoker			1.13	…
	Light Smoker			1.05	…
	Heavy Smoker			1.14 ++	…
	Heavy + Smoker			1.04	…
Parent Self-Rated Health	Excellent or Very Good			0.49 ***	0.49 ***
	Good			**1.00**	**1.00**
	Fair or Poor			1.55 ***	1.56 ***
Parent Weight Status	Underweight			0.82	…
	Normal			**1.00**	…
	Overweight			1.18 +++	…
	Obese			1.17 ++	…
	Morbidly Obese			1.99 ***	…
	Weight Unknown			1.00	…
Accumulation of Parental Health Behaviors§	No Negative Outcomes				**1.00**
	One Negative Outcome				1.25 *
	Two Negative Outcomes				1.42 **
	Three Negative Outcomes				1.26
	Pseudo R^2	0.0024	0.0262	0.0637	0.0619
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0010	0.0107	0.0258	0.0251
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0005	0.0054	0.0130	0.0126
	df	6	13	34	26
	-2LL	13894	13563	13040	13064
	Unweighted N	67903	67903	67903	67903

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "Does [child] have an impairment or health problem that limits his/her ability to crawl, walk, run, or play?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of in

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix D-I. Logistic Regression Estimates (Odds Ratios) for Child Having a Health Limitation,[†]
NHIS 1997-2003 Weighted.[¶]

Non Hispanic American Indians / Alaska Natives		**Baseline**	**Full Model**	**ALT Full Model**
Variables		(+) Child Characteristics	(+) Family / Parent Characteristics	(Δ) Parental Health Behaviors
	Constant	-2.49 ***	-1.75 ++	-1.54 ++
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	1.25	1.09	1.08
Age	0-4	0.14 ++	0.24 ++	0.20 ++
	5-11	0.22 +++	0.24 +++	0.20 +++
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	0.00 ***	0.00 ***	0.00 ***
	Low (1500-2499)	1.95	1.63	1.47
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	0.51	0.47	0.42
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		0.47	0.56
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.30 ++	0.29
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		1.43	1.78
Parent Household Composition	One Parent in Household		0.78	0.79
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance[‡]	Government Insurance		2.00	2.13
	Indian Health Service		1.10	1.04
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Smoking Status	Never Smoked		**1.00**	…
	Former / Current Smoker		0.85	…
Parent Weight Status	Underweight		3.81	…
	Normal		**1.00**	…
	Overweight		0.51	…
	Obese		0.47	…
	Unknown		0.00 ***	…
Parent Self-Rated Health	Excellent or Very Good		0.32	0.32
	Good		**1.00**	**1.00**
	Fair or Poor		2.82	2.24
Cumulation of Parental Health Behaviors[§]	No Negative Outcomes			**1.00**
	1 Negative Outcome			0.40
	2 to 3 Negative Outcomes			0.39
	Pseudo R^2	0.3703	0.6639	0.4911
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.1974	0.3259	0.2530
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.1041	0.1790	0.1357
	df	6	19	16
	-2LL	69	37	56
	Unweighted N	371	371	371

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "Does [child] have an impairment or health problem that limits his/her ability to crawl, walk, run, or play?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix D-II. Logistic Regression Estimates (Odds Ratios) for Child Having a Health Limitation,[†]
NHIS 1997-2003 Weighted.[¶]

Non Hispanic Whites		**Baseline**	**Full Model**	**ALT Full Model**
Variables		(+) Child Characteristics	(+) Family / Parent Characteristics	(Δ) Parental Health Behaviors
	Constant	-3.31 ***	-3.11 ***	-3.13 ***
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	0.93	0.93	0.93
Age	0-4	0.39 ***	0.44 ***	0.44 ***
	5-11	0.54 ***	0.58 ***	0.57 ***
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	5.67 ***	4.72 ***	4.64 ***
	Low (1500-2499)	1.99 ***	1.89 ***	1.88 ***
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	0.74 **	0.76 *	0.77 *
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		1.13	1.12
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.70 +++	0.70 *
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		0.97	0.98
Parent Household Composition	One Parent in Household		1.14	1.12
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance[‡]	No Insurance		1.01	1.00
	Government Insurance		1.78 ***	1.78 ***
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Smoking Status	Never Smoked		**1.00**	…
	Former / Current Smoker		1.13 ++	…
Parent Weight Status	Underweight		0.68	…
	Normal		**1.00**	…
	Overweight		1.20 ++	…
	Obese		1.22 ++	…
	Unknown		0.86	…
Parent Self-Rated Health	Excellent or Very Good		0.43 ***	0.43 ***
	Good		**1.00**	**1.00**
	Fair or Poor		1.45 *	1.45 *
Cumulation of Parental Health Behaviors[§]	No Negative Outcomes			**1.00**
	1 Negative Outcome			1.24 +++
	2 to 3 Negative Outcomes			1.38 *
	Pseudo R^2	0.0287	0.0656	0.0649
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0118	0.0268	0.0265
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0059	0.0135	0.0134
	df	6	19	16
	-2LL	7603	7314	7319
	Unweighted N	37,789	37,789	37,789

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "Does [child] have an impairment or health problem that limits his/her ability to crawl, walk, run, or play?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +)

Appendix D-III. Logistic Regression Estimates (Odds Ratios) for Child Having a Health Limitation,[†]
NHIS 1997-2003 Weighted.[¶]

Non Hispanic Blacks		**Baseline**	**Full Model**	**ALT Full Model**
Variables		(+) Child Characteristics	(+) Family / Parent Characteristics	(Δ) Parental Health Behaviors
	Constant	-3.63 ***	-4.30 ***	-4.30 ***
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	0.71 +++	0.70 +++	0.69 +++
Age	0-4	0.49 *	0.55 *	0.55 *
	5-11	0.96	1.04	1.03
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	3.72 ***	3.87 ***	3.85 ***
	Low (1500-2499)	1.83 +++	1.69 +++	1.64 ++
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	1.34	1.31	1.34
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		1.20	1.17
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		1.34	1.33
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		1.14	1.16
Parent Household Composition	One Parent in Household		1.07	1.08
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance[‡]	No Insurance		1.14	1.12
	Government Insurance		1.24	1.23
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Smoking Status	Never Smoked		**1.00**	…
	Former / Current Smoker		1.20	…
Parent Weight Status	Underweight		0.74	…
	Normal		**1.00**	…
	Overweight		1.05	…
	Obese		1.50 ++	…
	Unknown		1.42	…
Parent Self-Rated Health	Excellent or Very Good		0.84	0.82 **
	Good		**1.00**	**1.00**
	Fair or Poor		2.20 **	2.23
Cumulation of Parental Health Behaviors[§]	No Negative Outcomes			**1.00**
	1 Negative Outcome			1.21
	2 to 3 Negative Outcomes			1.84 +++
	Pseudo R^2	0.0189	0.0512	0.0514
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0083	0.0223	0.0224
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0042	0.0112	0.0113
	df	6	19	16
	-2LL	2068	2000	2000
	Unweighted N	9,570	9,570	9,570

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "Does [child] have an impairment or health problem that limits his/her ability to crawl, walk, run, or play?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix D-IV. Logistic Regression Estimates (Odds Ratios) for Child Having a Health Limitation,[†]
NHIS 1997-2003 Weighted.[¶]

Mexican Origin		**Baseline**	**Full Model**	**ALT Full Model**
Variables		(+) Child Characteristics	(+) Family / Parent Characteristics	(Δ) Parental Health Behaviors
	Constant	-3.80 ***	-3.93 ***	-4.05 ***
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	0.87	0.84	0.83
Age	0-4	0.62 +++	0.68 ++	0.67 ++
	5-11	0.78	0.83	0.82
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)	3.64 *	3.16 *	3.14 *
	Low (1500-2499)	1.02	0.95	0.95
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	0.91	0.87	0.88
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		1.39	1.39
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		1.00	0.99
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		2.20 *	2.20 *
Parent Household Composition	One Parent in Household		0.89	0.88
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance[‡]	No Insurance		0.67 ++	0.66 ++
	Government Insurance		0.99	0.98
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Smoking Status	Never Smoked		**1.00**	…
	Former / Current Smoker		0.98	…
Parent Weight Status	Underweight		0.35	…
	Normal		**1.00**	…
	Overweight		1.39	…
	Obese		1.39	…
	Unknown		1.01	…
Parent Self-Rated Health	Excellent or Very Good		0.44 ***	0.44 ***
	Good		**1.00**	**1.00**
	Fair or Poor		1.42	1.42
Cumulation of Parental Health Behaviors[§]	No Negative Outcomes			**1.00**
	1 Negative Outcome			1.60 ++
	2 to 3 Negative Outcomes			1.29
	Pseudo R^2	0.0078	0.0446	0.0451
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0026	0.0146	0.0148
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0013	0.0073	0.0074
	df	6	19	16
	-2LL	1756	1691	1690
	Unweighted N	10,732	10,732	10,732

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "Does [child] have an impairment or health problem that limits his/her ability to crawl, walk, run, or play?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix E. Logistic Regression Estimates (Odds Ratios) for Child Having at Least One Injury in the Past 3 Months,† NHIS 1997-2003 Weighted.¶

Variables		Baseline Race Only	Model 2 (+) Child Characteristics	Full Model (+) Family / Parent Characteristics	ALT Full Model (Δ) Parental Health Behaviors
	Constant	-3.37 ***	-2.92 ***	-2.93 ***	-2.91 ***
CHILD CHARACTERISTICS					
Race/Ethnicity	Non-Hispanic White	**1.00**	**1.00**	**1.00**	**1.00**
	Non-Hispanic AI/AN	1.04	1.02	0.99	0.97
	Non-Hispanic Black	0.49 ***	0.49 ***	0.48 ***	0.46 ***
	Mexican Origin	0.39 ***	0.41 ***	0.47 ***	0.44 ***
	Non-Hispanic Asian	0.14 ***	0.14 ***	0.16 ***	0.15 ***
	Non-Hispanic Other	0.70 +++	0.75 ++	0.72 ++	0.72 ++
	Other Hispanic	0.64 ***	0.66 **	0.70 *	0.67 *
Sex	Male		**1.00**	**1.00**	**1.00**
	Female		0.68 ***	0.68 ***	0.68 ***
Age	0-3		0.56 ***	0.60 ***	0.59
	4-7		0.56 ***	0.58 ***	0.57
	8-11		0.73 ***	0.75 **	0.75
	12-17		**1.00**	**1.00**	**1.00**
Birthweight	Very Low (<1500)		0.97	0.93	0.93
	Low (1500-2499)		1.04	1.02	1.03
	Normal (2500-3999)		**1.00**	**1.00**	**1.00**
	High (4000+)		1.06	1.05	1.05
FAMILY / PARENT CHARACTERISTICS					
Socioeconomic Factors					
Family Income to Poverty Ratio	Poor (< 1.00)			1.00	0.99
	Near Poor (1.00 - 1.99)			1.09	1.08
	Not Poor (2.00 +)			**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma			0.75 *	0.75 *
	High School Diploma or GED			0.80 *	0.80 *
	Some College			**1.00**	**1.00**
	College Degree			1.00	0.97
Parent Household Composition	One Parent in Household			1.17 +++	1.19 +++
	Both Parents in Household			**1.00**	**1.00**
Source of Insurance‡	No Insurance			0.92	0.91
	Government Insurance			0.91	0.92
	Private Insurance			**1.00**	**1.00**
Environmental Factors					
Parent Use of Alcohol	Does Not Drink			0.89 ++	…
	Light Drinker			**1.00**	…
	Moderate to Heavy Drinker			1.01	…
Parent Smoking Status	Never Smoked			**1.00**	…
	Former Smoker			1.19 +++	…
	Light Smoker			1.26 +++	…
	Heavy Smoker			1.39 ***	…
	Heavy + Smoker			1.39 *	…
Parent Self-Rated Health	Excellent or Very Good			0.86 +++	0.85 +++
	Good			**1.00**	**1.00**
	Fair or Poor			1.11	1.11
Parent Weight Status	Underweight			0.75	…
	Normal			**1.00**	…
	Overweight			1.12	…
	Obese			1.13 ++	…
	Morbidly Obese			1.42 +++	…
	Weight Unknown			0.92	…
Accumulation of Parental Health Behaviors§	No Negative Outcomes				**1.00**
	One Negative Outcome				1.16 +++
	Two Negative Outcomes				1.39 ***
	Three Negative Outcomes				1.64 *
	Pseudo R^2	0.0132	0.0239	0.0295	0.0279
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0066	0.0119	0.0147	0.0139
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0033	0.0060	0.0074	0.0070
	df	6	13	34	27
	-2LL	16809	16627	16532	16559
	Unweighted N	67903	67903	67903	67903

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "During the past three months, did you [or anyone in your family] have an injury where any part of [your/the] body was hurt?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix E-I. Logistic Regression Estimates (Odds Ratios) for Child Having at Least One Injury in the Past 3 Months,† NHIS 1997-2003 Weighted.¶

Non Hispanic American Indians / Alaska Natives		**Baseline**	**Full Model**	**ALT Full Model**
Variables		(+) Child Characteristics	(+) Family / Parent Characteristics	(Δ) Parental Health Behaviors
	Constant	-3.45 ***	-5.42 **	-6.15 *
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	1.78	0.94	2.02
Age	0-4	0.00 ***	0.00 ***	0.00 ***
	5-11	0.76	0.70	0.73
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Low (0-2499)	1.01	0.94	0.74
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	1.73	1.43	1.42
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		0.65	0.55
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.23	0.24
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		1.77	1.79
Parent Household Composition	One Parent in Household		1.26	1.18
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance‡	Government Insurance		0.44	0.48
	Indian Health Service		0.59	0.65
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Alcohol Use	Does Not Drink		**1.00**	…
	Currently Drinks		1.44	…
Parent Weight Status	Underweight		0.00 ***	…
	Normal		**1.00**	…
	Overweight		1.07	…
	Obese		1.23	…
	Unknown		0.00 ***	…
Parent Self-Rated Health	Excellent or Very Good		8.10 +++	9.52 +++
	Good		**1.00**	**1.00**
	Fair or Poor		24.57 *	25.88 *
Cumulation of Parental Health Behaviors§	No Negative Outcomes			**1.00**
	1 Negative Outcome			2.43
	2 to 3 Negative Outcomes			2.90
	Pseudo R^2	2.3880	4.0374	3.5918
	R^2 SAS	0.7599	0.9104	0.8830
	R^2 Cox & Snell	0.5100	0.7006	0.6580
	df	-154	-337	-287
	Unweighted N	371	371	371

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "During the past three months, did you [or anyone in your family] have an injury where any part of [your/the] body was hurt?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix E-II. Logistic Regression Estimates (Odds Ratios) for Child Having at Least One Injury in the Past 3 Months,† NHIS 1997-2003 Weighted.¶

Non Hispanic Whites

Variables		Baseline (+) Child Characteristics	Full Model (+) Family / Parent Characteristics	ALT Full Model (Δ) Parental Health Behaviors
	Constant	-2.86 ***	-3.08 ***	-3.09 ***
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	0.65 ***	0.65 ***	0.65 ***
Age	0-4	0.55 ***	0.57 ***	0.57 ***
	5-11	0.66 ***	0.68 ***	0.68 ***
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Low (0-2499)	1.12	1.10	1.09
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	0.93	0.93	0.94
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		1.06	1.04
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		1.14	1.11
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		1.22 *	1.26 *
Parent Household Composition	One Parent in Household		1.23 *	1.20 +++
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance‡	No Insurance		0.96	0.93
	Government Insurance		1.08	1.06
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Alcohol Use	Does Not Drink		**1.00**	…
	Currently Drinks		1.12	…
Parent Weight Status	Underweight		0.75	…
	Normal		**1.00**	…
	Overweight		1.17 ++	…
	Obese		1.19 ++	…
	Unknown		0.89	…
Parent Self-Rated Health	Excellent or Very Good		0.82 *	0.83 +++
	Good		**1.00**	**1.00**
	Fair or Poor		1.05	1.03
Cumulation of Parental Health Behaviors§	No Negative Outcomes			**1.00**
	1 Negative Outcome			1.16 ++
	2 to 3 Negative Outcomes			1.38 **
	Pseudo R^2	0.0118	0.0159	0.0158
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0068	0.0092	0.0091
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0034	0.0046	0.0046
	df	10860	10815	10816
	Unweighted N	37,789	37,789	37,789

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "During the past three months, did you [or anyone in your family] have an injury where any part of [your/the] body was hurt?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health p and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweigh

Appendix E-III. Logistic Regression Estimates (Odds Ratios) for Child Having at Least One Injury in the Past 3 Months,† NHIS 1997-2003 Weighted.¶

Non Hispanic Blacks

Variables		Baseline (+) Child Characteristics	Full Model (+) Family / Parent Characteristics	ALT Full Model (Δ) Parental Health Behaviors
	Constant	-3.54 ***	-3.88 ***	-3.85 ***
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	0.69 +++	0.69 +++	0.69 +++
Age	0-4	0.54 +++	0.62 ++	0.63 ++
	5-11	0.70 ++	0.75 ++	0.76
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Low (0-2499)	0.68	0.68	0.67
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	0.78	0.75	0.75
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		0.81	0.80
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.83	0.81
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		1.24	1.27
Parent Household Composition	One Parent in Household		1.35 ++	1.37 ++
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance‡	No Insurance		1.11	1.08
	Government Insurance		0.77	0.74
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Alcohol Use	Does Not Drink		**1.00**	…
	Currently Drinks		1.50 +++	…
Parent Weight Status	Underweight		0.91	…
	Normal		**1.00**	…
	Overweight		1.00	…
	Obese		1.01	…
	Unknown		1.33	…
Parent Self-Rated Health	Excellent or Very Good		0.80	0.83
	Good		**1.00**	**1.00**
	Fair or Poor		1.53 ++	1.48 ++
Cumulation of Parental Health Behaviors§	No Negative Outcomes			**1.00**
	1 Negative Outcome			1.17
	2 to 3 Negative Outcomes			1.59 ++
	Pseudo R^2	0.0105	0.0245	0.0225
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0035	0.0082	0.0075
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0018	0.0041	0.0038
	df	1586	1563	1566
	Unweighted N	9,570	9,570	9,570

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "During the past three months, did you [or anyone in your family] have an injury where any part of [your/the] body was hurt?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweight +).

Appendix E-IV. Logistic Regression Estimates (Odds Ratios) for Child Having at Least One Injury in the Past 3 Months,† NHIS 1997-2003 Weighted.¶

Mexican Origin

Variables		Baseline (+) Child Characteristics	Full Model (+) Family / Parent Characteristics	ALT Full Model (Δ) Parental Health Behaviors
	Constant	-3.99 ***	-3.63 ***	-3.85 ***
CHILD CHARACTERISTICS				
Sex	Male	**1.00**	**1.00**	**1.00**
	Female	0.62 +++	0.62 +++	0.62 +++
Age	0-4	0.76	0.80	0.81
	5-11	0.78	0.79	0.80
	12-17	**1.00**	**1.00**	**1.00**
Birthweight	Low (0-2499)	0.83	0.85	0.85
	Normal (2500-3499)	**1.00**	**1.00**	**1.00**
	High (3500+)	1.23	1.19	1.17
FAMILY / PARENT CHARACTERISTICS				
Socioeconomic Factors				
Family Income to Poverty Ratio	Poor (< 1.00)		0.72	0.71
	Not Poor (1.00+)		**1.00**	**1.00**
Parent Educational Attainment	No High School Diploma		0.76	0.75
	High School Diploma or GED		**1.00**	**1.00**
	At Least Some College		1.03	1.03
Parent Household Composition	One Parent in Household		0.92	0.94
	Both Parents in Household		**1.00**	**1.00**
Source of Insurance‡	No Insurance		0.94	0.93
	Government Insurance		0.79	0.78
	Private Insurance		**1.00**	**1.00**
Environmental Factors				
Parent Alcohol Use	Does Not Drink		**1.00**	…
	Currently Drinks		1.02	…
Parent Weight Status	Underweight		0.62	…
	Normal		**1.00**	…
	Overweight		0.92	…
	Obese		0.88	…
	Unknown		0.74	…
Parent Self-Rated Health	Excellent or Very Good		0.97	0.99
	Good		**1.00**	**1.00**
	Fair or Poor		0.34	1.32
Cumulation of Parental Health Behaviors§	No Negative Outcomes			**1.00**
	1 Negative Outcome			1.24
	2 to 3 Negative Outcomes			1.10
	Pseudo R^2	0.0082	0.0174	0.0176
	$\mathbf{R^2}_{\mathbf{SAS}}$	0.0023	0.0049	0.0050
	$\mathbf{R^2}_{\mathbf{Cox\ \&\ Snell}}$	0.0012	0.0025	0.0025
	df	1508	1494	1494
	Unweighted N	10,732	10,732	10,732

++ p<0.1, +++ p<.05, * p<.01, ** p<.001, ***p<.0001

† NHIS question, "During the past three months, did you [or anyone in your family] have an injury where any part of [your/the] body was hurt?"

¶ Universe is limited to children (from Child Sample) with corresponding parent (in Adult Sample) who was not pregnant at time of interview.

1.00 indicates reference category

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health p and other government health plans.

… Variable not included in alternative full model.

§ Negative parental health behaviors include drinking (moderate +), smoking (light +), and overweight/obesity (overweigh

Appendix F. Distribution of Cause and Place of Injury by Race-Ethnicity, NHIS 1997-2003, Weighted.

	non-Hispanic AI/AN	non-Hispanic White	non-Hispanic Black	Mexican Origin
Cause of Injury				
Transportation	7.60	11.54	18.67	14.66
Fire/burn/scald related	---	1.01	2.73	3.98
Fall	34.04	31.79	29.60	39.37
Poisoning	---	2.32	1.85	3.70
Overexertion/strenuous movements	2.72	6.21	2.95	2.35
Struck by object or person	20.10	24.14	23.86	17.28
Animal or insect bite	---	4.04	1.71	4.56
Cut/pierce	6.89	7.80	5.62	7.35
Machinery	---	0.10	---	---
Other	28.66	11.04	13.01	6.75
Place of Injury				
Inside home	---	24.16	25.54	27.96
Outside home	43.21	22.44	21.11	27.17
School	4.67	17.47	16.25	15.81
Child care center/preschool	---	1.12	1.44	---
Residential institution	---	0.16	0.87	---
Health care facility	---	0.22	0.22	---
Street/highway	4.32	8.17	15.88	8.62
Parking lot	---	0.71	1.70	1.79
Sport facility, ball field, playground	19.67	11.44	4.06	4.93
Trade and service areas	---	1.89	0.45	1.46
Farm	7.01	0.40	---	1.26
Park/recreation area	9.52	4.29	4.19	6.28
River/lake/stream/ocean	---	1.00	---	---
Industrial or construction area	---	0.31	---	---
Other public building	---	1.88	2.29	0.82
Other	11.59	3.77	6.00	3.91
REF, NA, DK, MISS	---	0.56	---	---

--- No cases reported.

Appendix G. SES by Utilization of Health Services by Race-Ethnicity, NHIS 1997-2003 Weighted.

	AI/AN			non-Hispanic White			non-Hispanic Black			Mexican Origin		
	% Never	% In the Last Year	% More than a Year Ago	% Never	% In the Last Year	% More than a Year Ago	% Never	% In the Last Year	% More than a Year Ago	% Never	% In the Last Year	% More than a Year Ago
Family Income to Poverty Ratio												
Poor (< 1.00)	0.37	82.67	16.96	0.93	89.71	9.36	0.60	88.62	10.79	4.18	78.71	17.11
Near Poor (1.00 - 1.99)	1.69	85.99	12.32	0.96	88.03	11.01	1.14	87.26	11.60	4.58	78.34	17.08
Not Poor (2.00 +)	1.08	87.19	11.73	0.62	92.53	6.85	1.02	90.86	8.12	2.51	86.41	11.09
Parental Educational Attainment												
No High School Diploma	1.44	84.91	13.65	1.32	86.72	11.97	1.40	87.07	11.53	5.43	75.21	19.36
High School Diploma or GED	0.37	86.13	13.50	0.85	89.53	9.62	0.87	87.63	11.50	2.10	85.60	12.30
Some College	0.66	85.76	13.58	0.59	92.05	7.36	0.85	90.29	8.86	1.99	88.54	9.48
College Degree	3.72	82.01	14.27	0.52	94.09	5.39	0.32	93.52	6.16	0.97	92.10	6.93
Source of Insurance‡												
No Insurance	0.00	44.32	55.68	2.04	81.29	16.67	1.56	78.42	20.02	8.11	65.71	26.19
Government Insurance	1.77	82.92	15.32	0.80	90.60	8.60	0.67	89.81	9.52	3.09	84.42	12.48
Indian Health Service§	1.59	86.16	12.25	…	…	…	…	…	…	…	…	…
Private Insurance	0.44	90.64	8.92	0.62	92.20	7.18	0.95	90.09	8.96	2.65	84.46	12.89

These rates reflect responses to "About how long has it been since anyone in the family last saw or talked to a doctor or other health care professional about [child's] health? Include doctors seen while {he/she} was a patient in a hospital."

‡ Government insurance coverage includes Medicare, Medicaid, Military, Indian Health Services, state-sponsored health plans, and other government health plans.

§ Represents only those AI/ANs who have IHS.

… Variable not included in crosstabulation.

Note: Distributions are shown by row categories such that each row per race-ethnic category adds to 100%.

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